ASSESSMENT OF CONTRIBUTORS OF METABOLIC SYNDROME AMONG OCCUPATIONAL DRIVERS

DISSERTATION SUBMITTED FOR

M.D., BRANCH-V (PHYSIOLOGY)

APRIL 2015

THE TAMILNADU

DR. M. G. R. MEDICAL UNIVERSITY
CHENNAI, TAMILNADU

BONAFIDE CERTIFICATE

This is to certify that the dissertation titled **"Assessment of Contributors of Metabolic Syndrome among Occupational Drivers''** is a bonafide record work done by **Dr.S.Sarumathy,** under my direct supervision and guidance, submitted to The Tamilnadu Dr.M.G.R. Medical University in partial fulfillment of University regulation for **M.D., Branch-V (Physiology).**

Dr.L.Santhanalakshmi,
M.D, D.G.O, MBA.,
Director and Professor,
Institute of Physiology,
MaduraiMedicalCollege,
Madurai.

Captain.Dr.B.Santhakumar,
M.Sc (F.Sc), M.D. (F.M), PGDMLE, DNB (F.M).,
Dean,
Madurai Medical College &
Govt. Rajaji Hospital,
Madurai.

DECLARATION

I, **DR.S.SARUMATHY,** solemnly declare that the dissertation titled ‘‘**ASSESSMENT OF CONTRIBUTORS OF METABOLIC SYNDROME AMONG OCCUPATIONAL DRIVERS’’** has been prepared by me. I also declare that this work was not submitted by me or any other, for any award, degree, diploma to any other University board either in India or abroad. This is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of **M.D degree Branch-V (Physiology)** to be held in **April-2015.**

Place: Madurai **Dr.S.SARUMATHY**

Date:

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I gratefully acknowledge all the subjects who co-operated to submit themselves for this study.

CONTENTS

PROFORMA

Name: Age:

Sex: Occupation:

H/O smoking:

No of cigarettes/day: For how many years:

H/O alcohol intake:

Nil/occasionally/weekly/monthly. For how many years:

Regular exercise:

Time spent/day: For how many years:

Diet: Vegetarian/Non vegetarian.

Duration of sleep/day:

Stress score as per PSS 10:

Duration of service:

Working hours/day:

	Duration	On treatment
H/O Diabetes:		
H/O Hypertension:		
H/O Hyperlipidemia:		

H/O Angina:

Family H/O Diabetes/Hypertension/Hyperlipedemia:

Medications if any:

Anthropometric measurements:

Ht (cm): Wt (kg): BMI(kg/m^2):

Waist circumference (cm): Waist hip ratio:

General Examination:

Consciousness: Orientation:

Comfortable at rest: Pallor:

Cyanosis: Clubbing:

Jaundice: Pedal oedema:

Thoracic/Spine deformity:

JVP: Generalized Lymphadenopathy:

Temperature (^{0}F): Respiratory rate/min:

Pulse rate/min: Blood pressure (mmHg):

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM :

ABDOMEN :

CENTRAL NERVOUS SYSTEM:

BIOCHEMICAL MEASUREMENTS:

Fasting blood glucose level (mg/dl):

Serum triglycerides (mg/dl):

Serum HDL (mg/dl):

மருத்துவப் பரிசோதனை முறைகளைப் பற்றி மருத்துவரிடம் தெரிந்து கொண்டேன். இதனை மேற்கொள்ள நான் முழுமனதுடன் சம்மதிக்கிறேன்.

0 =ஒருபோதும் இல்லை **1** = அனேகமாக இல்லை **2** =சிலவேளைகளில்
3 =பொதுவாக அடிக்கடி **4**= மிகஅடிக்கடி

1. கடந்த மாதம் ஏதிர்பாராத விதமாக நடந்த சம்பவங்களால் எத்தனை தடவைகள் கவலையுற்றீர்கள்?

   [0] [1] [2] [3] [4]

2. கடந்த மாதம் உங்கள் வாழ்க்கையிலுள்ள முக்கியமான விடயங்களை உங்களால் கட்டுப்படுத்த முடியாமல் இருந்ததாக எத்தனை தடவைகள் உணர்ந்தீர்கள்?

   [0] [1] [2] [3] [4]

3. கடந்த மாதம் எத்தனை தடவைகள் படபடப்புடன் கூடிய மனஉளைச்சலை உணர்ந்தீர்கள்?

   [0] [1] [2] [3] [4]

4. கடந்த மாதம் உங்கள் தனிப்பட்ட பிரச்சினைகளைச் சமாளிக்க உங்களுக்கு மனத்தைரியம் உள்ளதென எத்தனைதடவைகள் உணர்ந்தீர்கள்?

   [0] [1] [2] [3] [4]

5. கடந்த மாதம் நீங்கள் நினைத்ததுபோல் செயல்கள் யாவும் நடைபெற்றன என எத்தனை தடவைகள் உணர்ந்தீர்கள்?

   [0] [1] [2] [3] [4]

6. கடந்த மாதம் உங்களால் செய்யப்பட வேண்டியிருந்த எல்லா செயல்களையும உங்களால் ஈடுகொடுக்க முடியாமல் போய்விட்டதாக எத்தனை தடவைகள் உணர்ந்தீர்கள்?

   [0] [1] [2] [3] [4]

7. கடந்த மாதம் எத்தனை தடவைகள் உங்களக்கு எரிச்சலூட்டிய சொயல்களை கட்டுப்படுத்தக் கூடியதாக இருந்தது?

   [0] [1] [2] [3] [4]

8. கடந்த மாதம் எத்தனை பிரச்சினைக்குரிய செயல்களை முறியடித்ததாக உணர்ந்தீர்கள்?

   [0] [1] [2] [3] [4]

9. கடந்த மாதம் உங்களின் கட்டுப்பாட்டிற்கு அப்பாற்பட்ட செயல்களால் எத்தனை தடவைகள் கோபப்பட்டீர்கள்?

   [0] [1] [2] [3] [4]

10. கடந்தமாதம் உங்களால் வெற்றிகொள்ளமுடியாதுமென்மேலும் உயர்ந்துகொண்டிருந்தபிரச்சினைகளால் கஸ்ரப்பட்டதாகஎத்தனைதடவைகள் உணர்ந்தீர்கள்?

    [0] [1] [2] [3] [4]

Ref. No1864/E4/2/2014,

Govt. Rajaji Hospital,
Madurai.20. Dated:29.03.2014

Institutional Review Board / Independent Ethics Committee.
Capt. Dr.B. Santhakumar, M.D., (F.M.,) deanmdu@gmail.com
Dean, Madurai Medical College &
Govt Rajaji Hospital, Madurai 625020. **Convenor**

Sub: Establishment-Govt. Rajaji Hospital, Madurai-20-
Ethics committee-Meeting Minutes- for March 2014
Approved list - Regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held on 05.03.2014, Wednesday at 10.00 am to 12.00.noon at the Auditorium, Govt. Rajaji Hospital, Madurai. The following members of the committee have attended the meeting.

1.Dr.V. Nagarajan, M.D., D.M (Neuro) Ph: 0452-2629629 Cell.No 9843052029 nag9999@gmail.com	Professor of Neurology (Retired) D.No.72, Vakkil New Street, Simmakkal, Madurai -1	Chairman
2. Dr.Mohan Prasad , M.S M.Ch Cell.No.9843050822 (Oncology) drbkcmp@gmail.com	Professor & H.O.D of Surgical Oncology(Retired) D.No.32, West Avani Moola Street, Madurai -1	Member Secretary
3. Dr. Parameswari M.D (Pharmacology) Cell.No.9994026056 drparameswari@yahoo.com	Director of Pharmacology Madurai Medical College	Member
4. Dr.S. Vadivel Murugan, MD., (Gen.Medicine) Cell.No 9566543048 svadivelmurugan_2007@rediffmail.com	Professor& H.O.D of Medicine Madurai Medical College	Member
5. Dr.S. Meenakshi Sundaram, MS (Gen.Surgery) Cell.No 9842138031 drsundarms@gmail.com	Professor & H.O.D of Surgery Madurai Medical College	Member
6. Mrs. Mercy Immaculate Rubalatha, M.A., Med., Cell. No. 9367792650 lathadevadoss86@gmail.com	50/5, Corporation Officer's quarters, Gandhi Museum Road, Thamukam, Madurai-20	Member
7. Thiru..Pala. .Ramasamy , BA.,B.L., Cell.No 9842165127 palaramasamy2011@gmail.com	Advocate, D.No.72.Palam Station Road, Sellur, Madurai -2	Member
8. Thiru. P.K.M. Chelliah ,B.A Cell.No 9894349599 pkmandco@gmail.com	Businessman, 21 Jawahar Street, Gandhi Nagar, Madurai-20	Member

The following Projects was approved by the committee.

Name of P.G.	Course	Name of the Project	Remarks
Dr.S. Sarumathy, drsarumathy@gmail.com.	PG in MD., (Physiology) Madurai Medical College and Government Rajaji Hospital, Madurai.	Assessment of contributors to metabolic syndrome among Occupational Drivers.	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain it Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution or to Government.
2. She/He should inform the institution Ethical Committee, in case of any change of study procedure, site and investigation or guide.
3. She/He should not deviate the area of the work for which applied for Ethical clearance. She/He should inform the IEC immediately, in case of any adverse events or Serious adverse reactions.
4. She/He should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and if any Extension of time is required He/She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the work or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

Member Secretary **Chairman**
Ethical Committee

29.3.14
DEAN/Convenor
Govt. Rajaji Hospital,
Madurai- 20.

29/3/14

To
The above Applicant
-thro. Head of the Department concerned

To
Dr. S. Sarumathy.

1/4/14

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INTRODUCTION

Metabolic syndrome is a group of anthropological and biochemical abnormalities that predispose a person to cardiovascular disease, Type 2 Diabetes Mellitus and stroke. It is a group of interrelated abnormalities namely central obesity, raised blood pressure, high triglycerides, decreased levels of high-density lipoprotein cholesterol and elevated fasting blood glucose levels.

AIMS AND OBJECTIVES

To evaluate the occurrence of metabolic syndrome among occupational drivers and to assess the contributors of metabolic syndrome like physical activity, duration of driving, smoking, alcohol intake and stress level among occupational drivers.

METERIALS AND METHODS:

It is a cross sectional study, conducted in Jeyavilas bus depot, Madurai among 100 randomly selected male occupational drivers of age group between 25 to 60 years males who were drivers by occupation for > 5 years with minimum 8 hours of driving per day.

The study was initiated with the approval of Institutional ethical committee, Madurai Medical College, Madurai. After getting informed, written consent, a questionnaire is given for collecting the history. Basic cardiovascular and

anthropometric measurements are taken. Under strict aseptic precautions a blood sample of 3ml is collected after overnight fasting for at least 8 hours. Fasting blood glucose level, serum triglycerides and HDL analysis to be carried out using standard techniques.

RESULTS:

Statistical comparison between the measured variables carried out using Chi-Square test revealed significant association between increased BMI, physical inactivity, stress, smoking, increased duration of service with occurrence of metabolic syndrome.

CONCLUSION:

Drivers sit for long hours and walk less compared to the general public. In addition, physical activity involvement and physical demands during the driving are usually limited and insufficient to maintain physical fitness. The most effective measures to improve insulin sensitivity in metabolic syndrome affected individuals are exercise and weight loss. Both modalities are effective and can be additive in their ability to improve insulin action. Smokers should be advised to quit smoking. Change in the lifestyle is the best way in the prevention of metabolic syndrome.

Key words:

Metabolic syndrome, obesity, blood pressure, triglycerides, high-density lipoprotein cholesterol, fasting blood glucose, occupational drivers.

INTRODUCTION

The metabolic syndrome has received a great deal of attention in the last few decades. Metabolic syndrome is a group of anthropological and biochemical abnormalities that predispose a person to cardiovascular disease, Type 2 Diabetes Mellitus and stroke. It is a group of interrelated abnormalities namely central obesity, raised blood pressure, high triglycerides, decreased levels of high-density lipoprotein (HDL) cholesterol and elevated fasting glucose levels. This is a common metabolic disorder which increases in prevalence as the population becomes more obese.

Metabolic syndrome is associated with a 2-fold risk of cardiovascular disease and a 5-fold risk of diabetes. Persons with metabolic syndrome have a 30%–40% probability of developing Type 2 Diabetes Mellitus and/or cardiovascular disease within 20 years and it depends on the number of components present.

The World Health Organization has anticipated India as the diabetic capital of the world. At present the total number of people affected with diabetes worldwide is almost 150 million, with affected population of 25 million in India alone. By 2030, it is predicted that the number of people diagnosed with diabetes will reach about 366 million world wide, with the maximum number of cases in India.

Throughout the world, cardiovascular disease has become the most common cause of mortality. In 1990, the probable deaths due to cardiovascular disease was around 5.3 millions. At present, 8-9 millions, from the developing countries were affected with cardiovascular disease. By the year 2015, cardiovascular disease will be the most important contributor of mortality in India.

According to recent data in the Journal of the American Medical Association, it is estimated that 47 million people have metabolic syndrome. The incidence of this syndrome rises progressively as individuals begin to age, reaching a peak between the ages of 60 and 69 years and the prevalence increasing from 10% in the 30–39-year age group to 45% in the 60–69-year age group.

Based on data from the National Health and Nutrition Examination Survey (NHANES) III (1988-1994), nearly 24% of U.S. adults aged 20 years or older have metabolic syndrome. This rate has been raised to 27% in NHANES 1999-2000 and 34% in NHANES 2003-2006.

Similar to the western countries, the prevalence of metabolic syndrome in developing countries is also promptly increasing. A study by **Misra A and Khurana L** in 2008 showed that the situation is similar in

India with the recent data suggesting that upto one fourth and one third Indian adult population suffer from metabolic syndrome. **Chow et al., 2008** established a prevalence of metabolic syndrome of 26.9% in males and 18.4% in females in Southern India. This increase is observed regardless of the criteria used and it reflects the transition from a traditional to a Western lifestyle. These changes cause significant effects on body composition often causing a rise in BMI, generalized and abdominal obesity, an increase in dyslipidemia and Type 2 Diabetes Mellitus. Intrauterine and early postnatal undernutrition have been suggested as one of the important causes of development of metabolic syndrome **Misra A et al., 2008**. In general, the International Diabetes Federation estimates that one-quarter of the world's adult population has metabolic syndrome (IDF 2006).

The study conducted by **Rajeev Gupta et al., 2012** in Jaipur, showed the prevalence of metabolic syndrome was 25.1% in males and 22% in females in urban population. Occurrence of diabetes and hypertension was more in men, low HDL cholesterol levels and abdominal obesity was more in women.

It is obvious that occupational drivers are associated with considerable changes in lifestyle habits. Occupational drivers are prone to develop

metabolic syndrome because their working environment is characterized by numerous stress factors such as lack of physical activity due to working in a stationary position, disruption in diet and irregular sleep pattern.The related impacts are not only harmful for driver's health but also may endanger others, because any health issue that affects drivers may result in an increased risk for road accidents. The aim of the present study is to evaluate the contributors of metabolic syndrome among occupational drivers in Madurai district.

AIMS AND OBJECTIVES

1. To assess the contributors of metabolic syndrome among occupational drivers.
2. To evaluate the occurrence of metabolic syndrome among occupational drivers.
3. To study the correlation of BMI, physical activity and metabolic syndrome.
4. To assess the relationship between duration of driving with occurrence of metabolic syndrome.
5. To study the effect of smoking and alcohol intake with occurrence of metabolic syndrome.
6. To analyze the link between stress level with occurrence of metabolic syndrome.

REVIEW OF LITERATURE

HISTORY OF METABOLIC SYNDROME

In 1920s, it was proposed that diabetes was associated with various risk factors but the term "metabolic syndrome" was first used since 1950 and only in late 1970 onwards, it came into common practice.

In 1947,**Dr. Jean Vague**, the Marseilles physician, found that abdominal obesity predisposed to gout, atherosclerosis, diabetes and calculi.

Crepaldi, Avogaro and their colleagues performed a study on obese patients with marked hypertriglyceridemia and diabetes. All of them were put on a low carbohydrate diet and they showed improvement.

In 1988, **Gerald M.Reaven**, an endocrinologist physician from Stanford University was the one who interpreted the association of diabetes, obesity, dyslipidemia and arterial hypertension by their pathogenic relationship with the peripheral insulin-resistance. He named this association **"X syndrome"**, the name underlining the doubtfulness of emitting an apparently new concept. The insulin resistance and the compensatory

hyperinsulinism were associated with each component of the metabolic syndrome, proposing thus a physio-pathological connection between them.

The X syndrome was renamed as **"The Deadly Quartet"** by **Kaplan** in 1989. It was again renamed as **"The insulin resistance syndrome"** in 1992.

Afterwards, it was found out that the spectrum of metabolic disturbances is greater. **Zimmet** expressed the **"plus X syndrome"** establishing the association with hyperuricaemia, sedentariness and old age.

On 1998, in the Chronic Fatigue Syndrome Conference, **Dr Allen E. Gale**, a physician, presented a case report in Australia and proposed the term **CHAOS**, which is an abbreviation for Coronary artery disease, Hypertension, Atherosclerosis, Obesity and Stroke.

CRITERIA FOR METABOLIC SYNDROME

At present, four sets of criteria are most commonly used for defining the metabolic syndrome.

WHO---CRITERIA:

The WHO definition,1998was the first to link the key components of insulin resistance, obesity, dyslipidemia and hypertension. The definition mandates the presence of insulin resistance, in addition any two of the following:

- Central obesity-Body mass index >30 kg/m^2 or

  Waist:Hip ratio> 0.85 (female), > 0.90 (male)

- Dyslipidaemia-HDL $\leq$ 0.9 mmol/L (male) $\leq$ 1.0 mmol/L (female)

  Triglycerides $\geq$ 1.695 mmol/L.

- Blood pressure- $\geq$ 140/90 mmHg.
- Microalbuminuria-albumin : creatinine ratio $\geq$ 30 mg/g or

  urinary albumin excretion ratio $\geq$ 20 µg/min.

NCEP:ATPIII 2001-CRITERIA:

In 2001, the National Cholesterol Education Programme (NCEP) Adult Treatment Panel III (ATP III) framed a definition for the metabolic syndrome (National Cholesterol Education Programme, 2002), it was updated by the American Heart Association and the National Heart Lung and Blood Institute in 2005 **Grundy et al., 2004**. According to it, metabolic syndrome is present if three or more of the following five criteria are met:

- Blood pressure: systolic ≥ 130 or diastolic ≥ 85 mmHg .
- Fasting triglyceride (TG) level : Triglycerides $\geq$150mg/dl.
- Waist circumference : >40 inches (male), >35 inches (female)
- Fasting plasma glucose: ≥ 110mg/dl.
- Fasting HDL level: <50 mg/dl (male), < 40 mg/dl (female).

It is one of the most widely used criteria of metabolic syndrome. It includes the key features such as hyperglycemia/insulin resistance, visceral obesity, atherogenic dyslipidemia and hypertension. It makes use of the measurements and laboratory results which would be readily available to physicians, allowing its clinical and epidemiological application. Importantly, it does not necessitate that any specific criterion be met.

IDF-CRITERIA :

In 2005, the International Diabetes Foundation (IDF) has put out a new criteria for metabolic syndrome **Zimmet et al., 2007**.

The IDF definition of the metabolic syndrome is.

Central obesity PLUS any two of the following:

- Elevated fasting plasma glucose (FPG): >100 mg/dl or a known case of Type 2 Diabetes Mellitus.
- Fasting triglycerides >150mg/dl or specific medication.
- HDL < 50 mg/dl and < 40 mg/dl for women and men respectively.
- Blood pressure >85 mmHg diastolic or >130 mmHg systolic or a known case of hypertension.

Waist circumference with geography – specific values for defining Central obesity.

Central Adiposity by IDF Criteria:

Men	Women	Ethnicity
≥85cm	≥ 90cm	Japanese.
≥90cm	≥80cm	Central American, Chinese and South Asian.
≥94cm	≥80cm	Middle Eastern, Eastern, Europid and Sub-Saharan African.

The WHO and IDF definitions are limited by their applicability and clinical acceptability. The most accepted definition is the NCEP-ATP III one because of his simplicity.

MODIFIED NCEP ATPIII- CRITERIA FOR THE METABOLIC SYNDROME:

In 2005, the American Heart Association and the National Heart, Lung and Blood Institute (AHA / NHLBI) conducted a revision of the metabolic syndrome diagnostic criteria - modified NCEP ATPIII criteria.

The important changes in the modified AHA/NHLBI definition, ATPIII 2005**Grundy et al., 2005** include:

(i) Addressing the ethnic-specific variation in central obesity by using the WHO recommendations for waist circumference: 90 cm in Asian men, 80 cm in Asian women.

(ii) Decreasing the threshold for impaired fasting glucose to $\geq$ 100mg/dl, in accordance with the American Diabetes Association (ADA) revised definition **Genuth et al., 2003**.

(iii) Permitting individual components to be counted abnormal if patients are receiving drug treatment for these conditions.

By making these difference the predictive ability to diagnose metabolic syndrome has become highest with modified NCEP-ATP III.

SIGNS AND SYMPTOMS

- Central obesity (otherwise referred as visceral, apple-shaped or android type of adiposity).
- Elevated blood pressure.
- Fasting hyperglycemia- impaired fasting glucose or impaired glucose tolerance, Type 2 Diabetes Mellitus.
- Elevated triglycerides.
- Reduced High-density lipoprotein cholesterol.

PATHOPHYSIOLOGY

Metabolic Syndrome is the result of complex interplay between genetic and environmental factors. Understanding in detail about the pathophysiology of this syndrome is important in order to identify people at risk of developing cardiovascular disease which will help in early intervention for prevention **Lann D, LeRoith D 2007**.

Functional aberrations seen in metabolic Syndrome are:

1. Insulin resistance
2. Dyslipidemia
3. Central obesity
4. Hypertension
5. Proinflammatory state
6. Prothrombotic state.

1.INSULIN RESISTANCE

The metabolic syndrome is also known as the insulin resistance syndrome, since it has been postulated that insulin resistance is the key mechanism responsible for the metabolic abnormalities of the syndrome **Lann D, LeRoith D 2007**. Insulin resistance has been defined as a defect in

insulin action that results in hyperinsulinaemia which is necessary to sustain the euglycaemic state. Concept of insulin resistance provides a conceptual framework which places a substantial number of apparently unrelated biological events into a pathophysiological construct.

Insulin is synthesized by the pancreas in response to hyperglycemia and it stimulates glucose use differently in different tissues.

Effect of insulin on various tissues are:

- In the skeletal muscle and adipose tissue ⟶ stimulates glucose uptake by translocation of the GLUT4 glucose transporter to the surface of the cell.
- In the skeletal muscle and liver ⟶ stimulates the synthesis of glycogen from glucose and they inhibits glycogenolysis.
- In the liver⟶ decreases hepatic gluconeogenesis, thus preventing an influx of more glucose into the bloodstream.
- In adipose tissue ⟶ inhibits fat breakdown and stimulates glucose uptake.

The net effect of all of these mechanism is to

1.Increase glucose uptake,

2.Reduce circulating glucose levels and

3.Increase the conversion of glucose into the storage molecules, glycogen or fat **Kim et al., 2006**.

Normally fasting insulin level is between 5-15µU/ml. But in Insulin resistance it is about >15 µU/ml. In insulin resistance, adipose tissue, muscle and liver do not respond to insulin, hence the blood glucose levels remain high, leading to pathology.

When insulin signalling occurs, insulin binds to the insulin receptor, which is a ligand-activated tyrosine kinase. Binding of insulin produces tyrosine phosphorylation of downstream substrates and activation of two parallel pathways:

1.The phosphoinositide 3-kinase (PI3K) pathway and

2.The mitogen-activated protein (MAP) kinase pathway.

1. Activation of PI3K, leads to activation of the 3-phosphoinositide-dependent protein kinase 1 (PDK1) kinase and Akt kinase. The PI3K-Akt pathway help in many of the metabolic effects of insulin.

- In vascular endothelial cells, it causes phosphorylation and activation of endothelial nitric oxide synthase (eNOS).
- In skeletal muscle and adipose tissue, it stimulates translocation of GLUT4 glucose transporter to the surface of the cell leading to increased glucose uptake.

2. Similarly, activation of MAP kinase pathway is responsible for

- Endothelin-1(ET-1) production which leads to vasoconstriction.
- Expression of the vascular cell adhesion molecules (VCAM-1) and E-selectin resulting in more leukocyte-endothelial interactions and
- Mitogenic action on vascular smooth muscle cells.

In insulin resistance, the PI3K-Akt pathway is affected, while the MAP kinase pathway remains unaffected. This leads to a change in the balance between these two parallel pathways.

Since PI3K-Akt pathway is affected, it leads to a decrease in endothelial nitric oxide (NO) production causing endothelial dysfunction. This also leads to a reduction in GLUT4 translocation which leads to decreased glucose uptake in skeletal muscle and fat. By contrast, the MAP

kinase pathway is unaffected, hence there is a continued production of ET-1, expression of VCAM-1 and mitogenic stimulus to vascular smooth muscle cells. By these mechanisms, insulin resistance results in vascular abnormalities which predisposes to atherosclerosis.

Insulin increases local blood flow in tissues by the activation of eNOS **Kim et al., 2006; Jonk et al., 2007**. This leads to two separable effects

1. Capillary recruitment occurs within minutes.
2. Dilation of the larger-resistance vessels increases overall perfusion between 30 minutes and 2 hours.

These two effects contribute to vasodilation and increased delivery of glucose and insulin to tissues. The vascular effects of insulin couples with glucose homeostasis and contribute to glucose metabolism at physiological concentrations of insulin. Thus, insulin acts coordinately on peripheral glucose use, vascular tone and blood flow. Therefore common mechanisms that contribute to insulin resistance can also cause vascular dysfunction, hyperglycemia, etc.

The linked concepts of metabolic syndrome helped us by providing a simple construct to characterize many types of patients who the physicians see daily and to help them identify people at risk **Yehuda H 2009**.

2.DYSLIPIDEMIA

A major contributor to the development of insulin resistance is an excess of free fatty acids (FFA), released from an expanded adipose tissue. Excessive release of free fatty acids from adipose tissue into plasma and increased plasma free fatty acids concentration can impair insulin-mediated glucose uptake in muscle and suppress hepatic glucose production. Increased level of circulating glucose increases insulin secretion resulting in hyperinsulinemia.

In the liver, FFAs acts as a substrate for synthesis of triglycerides (TGs). FFAs also stabilize the synthesis of apoB, which is a chief lipoprotein of very-low-density lipoprotein (VLDL) particles, this results in increased production of VLDL. VLDL in turn is metabolized to remnant lipoproteins and small dense LDL, both of which can stimulate atheroma formation.

The TGs in VLDL are transferred to HDL by the cholesterol ester transport protein (CETP) in exchange for cholesteryl esters. This results in TG-enriched HDL and cholesteryl ester-enriched VLDL particles. The TG-enriched HDL is a better substrate for hepatic lipase, hence it is rapidly cleared from the circulation leaving behind very few HDL particles to contribute in reverse cholesterol transport from the vasculature. Ultimately

resulting in increased plasma triglyceride and LDL levels and decreased plasma HDL level.

Insulin is the major physiological regulator of adipose tissue lipolytic activity by inhibiting lipolysis. Lipolysis of adipose tissue triglycerides is the main source of plasma FFA. In the case of insulin resistance, the rate of lipolysis is increased producing more amount of fatty acids which in turn further inhibit the antilipolytic action of insulin thereby producing additional lipolysis.

In skeletal muscle, the mechanism responsible for free fatty acids-induced insulin resistance is alteration in intracellular insulin signalling and impaired insulin-mediated glucose uptake. An acute increase in plasma free fatty acids concentrations from approximately 400 μM (normal basal concentration) to approximately 800 μM (concentration during short-term fasting) causes a marked increase in intramyocellular fatty acid metabolites which include long-chain fatty acyl-CoA and diacylglycerol. These metabolites are potent allosteric activators of protein kinase C which phosphorylates serine/threonine sites of insulin receptor substrate-1. This results in inhibition of insulin's ability to activate phosphoinositide 3-kinase

activity and decreases downstream events including translocation of GLUT-4 from the cytoplasm to the surface of the cell needed for glucose transport.

Defective skeletal muscle mitochondrial function has been identified in persons who have insulin resistance and are at increased risk for developing Type 2 Diabetes Mellitus. Impaired mitochondrial fatty acid oxidation increases the intracellular accumulation of fatty acids leading to impaired insulin action.

Development of atherosclerotic plaque:

Fatty streak is the earliest lesion of atherosclerosis made up of lipid laden foam cells, derived mainly from circulating monocytes and few smooth muscle cells. Foam cells represent circulating monocytes, that penetrate between endothelial cells, entered the intima and taken up lipoproteins and consequent storage of cholesterol esters in multiple droplets.

In diabetes, hyperglycemia and hypercholesterolemia activates and induces expression of specific adhesion molecules like VCAM, ICAM, E-SELECTIN etc on the endothelial cells.

It also influences the behaviour of circulating monocytes increasing its adhesion to the endothelium. Monocytes adhere and penetrate in response to chemotactic factors, like MCP-1 which is unregulated in Diabetes. The extracellular lipid begins to accumulate in the intima which often associates with proteoglycans of extracellular matrix. Sequestration within the intima separates lipoproteins from plasma antioxidants and favours oxidative alteration.

Evolution of fibro fatty streak

The next step is accumulation of lipids. Hyperglycemia in Diabetes Mellitus causes glycation of LDL. Glycation of LDL apoB occurs mainly in lysine residues in the LDL receptor binding domain, essential for specific recognition of LDL by the LDL receptors. Thus it leads to impairment of LDL receptor mediated uptake which inturn leads to decreased clearance of LDL.

Glycation confers increased susceptibility of LDL to oxidative modification. Advanced glycation of an amine containing phospholipid component of LDL is accompanied by progressive oxidative modification of unsaturated fatty acid residues. LDL glycation enhances its uptake by

aortic intimal cells and monocyte derived macrophages stimulating the development of foam cells.

Glycation and oxidation are closely related and mutually accelerate each other. Product of combined glycation and oxidation of LDL (glyoxidation) is more atherogenic than glycated or oxidized LDL alone.

Dyslipidemia in Diabetes Mellitus (↓ HDL &↑ TGL) reduces reverse cholesterol transport and increases the formation of small dense LDL, which is more prone for oxidation. Small dense LDL enters the arterial intima more easily and binds more readily to the proteoglycans, through specific sequence of apoB, than the larger fraction. Binding of LDL in the arterial intima increases the dwelling time and provides opportunity for oxidation of LDL lipids.

Oxidised LDL has several pro atherogenic properties including the rapid uptake by macropages to form foam cells, chemoattraction for circulating monocytes, promotion of the differentiation of circulating monocytes into tissue macrophages and inhibition of the mobility of resident macrophages. It is also cytotoxic to several types of cells and immunogenic.

Macrophage foam cells, leucocytes and resident vascular wall cells can secrete inflammatory cytokines and growth factors (TNFα,IL-1,MCP-

1,PDGF,FGF,TGF,etc) that amplify leucocyte recruitment and causes smooth muscle cell migration and proliferation. Thus conversion of fatty streak to fibrous plaque occurs by accumulating vascular smooth muscle cells and accumulating a complex extracellular matrix that consists of proteoglycans, collagen and elastin.

Fibrous cap formation:

The fibrous material that separates the plaque core from the luminal surface of the vessel is termed as fibrous cap. The fibrous cap provides a barrier to thrombosis by separating the pool of tissue factor from the FactorVII in the blood. Increased matrix metalloproteinase activity and macrophage in Diabetes Mellitus may shift the proteolytic balance within the cap leading to degradation of extracellular matrix and weakening of its structural integrity, thus increasing susceptibility to rupture or erosion.

Atherothrombosis:

Disruption of plaque surface allows the tissue factor to bind circulating factorVII in blood resulting in the generation of active factorVIIa and formation of tissue factor-factorVIIa complex, leading to activation of coagulation cascade. Elevated levels of tissue factor in vascular tissue, predispose the activation of coagulation cascade and increased arterial

expression of Plasminogen activator inhibitor -1 in diabetes may impair the dissolution of nascent thrombi and this may lead to occlusive thrombi and thereby can cause acute coronary syndrome. Thus oxidation of LDL plays major role in development of atherosclerosis in Diabetes Mellitus.

3.CENTRAL OBESITY

According to the new criteria of IDF, Metabolic syndrome can also be called as central obesity syndrome **Gary 2006**. Central obesity is a high risk factor for cardiovascular disease. Central obesity is more metabolically active than peripheral fat. Recently, studies have proposed that central adiposity precedes the development of the other components of metabolic syndrome and weight reduction at that point could be the best way to prevent it. **Pladevall et al., 2006, Steele et al., 2008** recommends that waist circumference to be measured regularly to assess individuals for increased risk for insulin resistance and to target individuals for health promotion interventions. Though insulin resistance is the major factor for the development of metabolic syndrome, it is the obesity that provides the connection between the insulin-resistant, dyslipidemic and hypertensive factors **Wingard et al.,1996**.

Body fat distribution are of two types

1. 90 % of the fat is Subcutaneous fat.
2. Remaining 10 % of fat is Visceral fat.

Subcutaneous fat is the kind felt when the skin is pinched. It can be measured using body fat calipers, which gives a rough estimate of total body adiposity.

Visceral fat lies beneath the abdominal muscles and can only be detected by MRI. Since, MRI scans are not a cheap procedure it is not recommended as a diagnosis tool for diabetes risk. Research has shown that the size of our belly is a relatively reliable indicator of the health risks linked to visceral fat, which can be assessed by waistline measurement. Since around 10% of our total fat is likely to be stored as visceral fat, therefore if a person carries higher amounts of body fat than the recommended level, the person is likely to have more visceral fat than is healthy.

Visceral fat is more dangerous than the subcutaneous fat. It is found that stress has a significant effect on where fat is stored in our body. Researchers have shown that cortisol, the stress hormone, significantly rises the storage of visceral fat.

Visceral fat releases free fatty acids directly into portal circulation, carrying blood directly to the liver. It also gets accumulated in the pancreas, heart and other organs. This leads to organ dysfunction, impaired insulin regulation, blood glucose and cholesterol level as well as abnormal heart functions. This is known as lipotoxicity **(Havard College 2006).**

Adipose tissue produces several inflammatory cytokines (adipokines), which induces insulin resistance, and adiponectin which can rise the insulin sensitivity **Kershaw and Flier, 2004**. The adipokines include tumour necrosis factor α (TNFα) and interleukin-6 (IL-6), which are proinflammatory and induce insulin resistance and vascular dysfunction. By contrast, adiponectin is a protective adipokine that increases insulin sensitivity in the liver, reduces hepatic glucose production and increases skeletal muscle glucose and fatty acid oxidation. Adiponectin levels are reduced in obesity, Type 2 Diabetes Mellitus and metabolic syndrome.

4.HYPERTENSION

One of the symptoms of metabolic syndrome is hypertension. It is a silent symptom which may remain undetected for long period. It is an important risk factor for development of cardiovascular disease. The relation

between insulin resistance and hypertension is well established. Several different mechanisms are proposed.

Insulin is a vasodilator when given intravenously with secondary effects on sodium reabsorption in the kidney. In case of insulin resistance, the vasodilatory effect of insulin is lost but the sodium reabsorption by the kidneys is preserved.

Fatty acids themselves can cause relative vasoconstriction.

Hyperinsulinaemia leads to increased sympathetic nervous system (SNS) activity, resulting in the development of hypertension.

Hypertension is a chronic condition of concern, due to its role in the causation of cardiovascular mortality which accounts for 20-50 % of all deaths. Hypertension is one of the common disease, leading to many life threatening complications including, renal failure, cerebrovascular accidents and atherosclerotic coronary artery disease. But, the effects of hypertension are not diagnosed in its early stages. About 85% of individuals affected with metabolic syndrome will have elevated blood pressure.

Criteria for diagnosis of Hypertension

Systolic BP mmHg	Diastolic BP mmHg	Category
Less than 120 and	Less than 80	Normal
120 – 139 or	80 – 89	Prehypertension
140 – 159 or	90 – 99	Stage 1 Hypertension.
≥ 160 or	≥ 100	Stage 2 Hypertension.

Hypertension is divided into primary (essential) and secondary. Among all the cases of hypertension, 90% belongs to primary hypertension. Secondary hypertension is due to congenital narrowing of aorta, tumors of adrenal glands, diseases of kidney, etc. Altogether these diseases were estimated to account for 10% of cases of hypertension.

5.PROINFLAMMATORY STATE

Yudkin et al., 1999 noted that low-grade inflammation is associated with insulin resistance. The origin of the inflammatory state and endothelial dysfunction are adipocyte-generated inflammatory cytokines, which correlate strongly with insulin resistance. Circulating signal molecules from fat include FFAs, adiponectin, IL-6, resistin, leptin and TNF-α. Studies shows associations of levels of C-reactive protein and interleukin-6 with measures of obesity and of chronic infection as their presumed determinants.

Studies also related levels of C-reactive protein and interleukin-6 to markers of the insulin resistance syndrome and of endothelial dysfunction. Metabolic syndrome and obesity are a kind of stress that leads to activation of inflammatory pathways. In fact the inflammation is low grade chronic inflammation. Researchers have named this inflammatory state as ―metainflammation, which means metabolically triggered inflammation.

6.PROTHROMBOTIC STATE

Increased levels of plasma plasminogen activator inhibitor (PAI)-1 and fibrinogen are also associated with the metabolic syndrome. Thus, prothrombotic and proinflammatory states are interconnected.

The study of plasminogen activator inhibitor-1 helps in better understanding of association between hemostatic markers and metabolic syndrome. A study was conducted by **Aso et al., 2005** to determine whether plasma concentrations of thrombin-activatable fibrinolysis inhibitor (TAFI) in patients with Type 2 Diabetes Mellitus were associated with components of metabolic syndrome, including high-sensitivity C-reactive protein (hs-CRP), plasminogen activator inhibitor (PAI)-1 and LDL cholesterol. The result indicated positive correlation between LDL cholesterol and plasma TAFI with Type 2 Diabetes Mellitus. Co-existence of metabolic syndrome and hypercholesterolemia accelerates inflammation and elevated TAFI and

PAI-1, inhibits fibrinolysis. PAI-1 is an important risk factor for metabolic syndrome. Three other biomarkers, CRP, IL6 and fibrinogen are also associated with the metabolic syndrome cluster. These 4 biomarkers contribute in the metabolic syndrome risk assessment **Kraja AT et al., 2007**.

RISK FACTORS

The metabolic syndrome also has multiple risk factors such as obesity, physical inactivity, unhealthy diet, ageing, disruption of sleep, smoking and stress.

1. OBESITY

Central adiposity is a key feature of the metabolic syndrome, which implies that the syndrome's prevalence is driven by the strong relationship between waist circumference and increasing adiposity. Even then the patients of normal weight can also be insulin resistant. Those are called metabolically obese, normal-weight individuals. These persons have increased amount of visceral adipose tissue. Effective weight reduction is known to improve individual components of the metabolic syndrome, while weight gain during adulthood is known to exacerbate individual components of the metabolic syndrome.

Obesity is a chronic condition characterized by the presence of abnormal or excessive body fat. Obesity also increases the chance of various chronic diseases like cardiovascular disease, stroke, Type 2Diabetes Mellitus and few types of cancer **Haslam D 2005**. At an individual level, excessive

body fat results when energy intake exceeds energy expenditure **Lau D et al., 2007.**

The odds of having metabolic syndrome increase with increasing levels of BMI in men and women. The metabolic syndrome was found in 4.6%, 22.4%, and 59.6% of normal weight, overweight and obese men respectively and in 6.2%, 28.1% and 50.0% of normal weight, overweight and obese women respectively based on the NHANES III. Thus most persons with the metabolic syndrome are overweight or obese, suggesting that obesity in conjunction with genetic aspects of susceptibility may link the components of the metabolic syndrome.

Sedentary occupations with high levels of sitting time and lower demand for physical activity have been associated with weight gain and obesity.

Assessment of Obesity

Obesity is usually easily identified at first sight. It is diagnosed by physical appearance which is referred to as "eyeball test".

In clinical medicine, currently waist circumference, waist hip ratio and BMI are the commonly used parameters for assessing obesity. The introduction of measuring fat cells has opened up a new field in obesity research.

Body Mass Index (BMI)

BMI is the most often used method for estimating obesity. When BMI is more than 30, the individual is considered to be obese.

BMI is calculated by using the Quetelet's Index.

BMI = Weight (Kg)/ Height (m^2).

For adults, overweight and obesity are defined as a BMI of 25 kg/m^2 to 29.9 kg/m^2 and 30 kg/m^2or higher, respectively.

Waist Circumference and Waist-to-Hip Ratio (WHR)

The mid point between iliac crest and the lowest rib was measured with an inch tape in standing posture for assessing the waist circumference. In the gluteal region the widest part was measured as Hip circumference and Waist Hip Ratio was calculated.

The distribution of fat is better assessed by Waist Hip Ratio. The risk factors for chronic diseases like coronary artery disease is associated with an increase in waist circumference. The total body fat and intra-abdominal fat mass is better assessed by waist circumference which is an easy and convenient measure. It is not related to height, but is better correlated with change in Waist Hip Ratio and Body Mass Index.

There are two types of body shape that are related to the health –

- Apple shaped body
- Pear shaped body

Body shape can be determined by using the Waist Hip Ratio. It is important because it indicates the fat distribution in your body. The body shape is determined by a number of factors. These include genes, diet and life style.

People with apple-shaped bodies carry more weight around their abdomen that is, they have more amount of visceral fat, hence this can be associated with a greater risk of heart disease, diabetes and stroke.

Those with pear-shaped body have a narrower waist and carry more weight around their hips and thigh, it indicates a lower metabolic risk.

A Waist Hip Ratio of 0.8 or above indicates an apple shaped body. A ratio of below 0.8 indicates a pear shaped body.

The prevalence of obesity among adults in the year 2000 was found to be 55% out of which 24% were females and 21% were males. Throughout the world, there are about 250 million obese individuals.

In United States alone, 30,000 deaths were related to over weight and obesity.

In a study conducted at Mangalore, around 29.7% of people were affected with metabolic syndrome and the prevalence was more in female subjects which is attributed by their increased waist circumference.

2. SEDENTARY LIFESTYLE

Physical inactivity is a predictor of cardiovascular disease and related mortality rate. Many components of the metabolic syndrome are associated with a sedentary lifestyle including increased adipose tissue (mainly visceral), decreased HDL cholesterol and increased triglycerides, blood pressure and blood glucose in the genetically susceptible individuals. Individuals who watched television or videos or used the computer >4 hours daily have a twofold increased risk of the metabolic syndrome than those who carried out those behaviors for <1 hour daily. Regular and sustained physical activity positively influences individual components of the metabolic syndrome.

Physical activity has been defined as any bodily movement produced by skeletal muscles that result in energy expenditure beyond resting expenditure **Thompson P et al., 2003**. Many previous studies have

demonstrated that physical activity is associated with reduced risk of the metabolic syndrome and physical inactivity may be an important modifiable risk factor in the etiology of the metabolic syndrome **Torjesen P et al., 1997**. While the metabolic syndrome has increased in the past few decades, the amount of physical activity and energy expenditure has declined during this time. Thus, individuals with a lack of physical activity may be at risk of metabolic syndrome.

Ekelund and colleagues studied the prospective associations between energy expenditure due to physical activity and the progression toward the metabolic syndrome in 605 middle aged men and women who do not suffer from metabolic syndrome over a 5.6 year follow-up period. The measure of physical activity in this study was based on the amount of energy expenditure above resting level including all types of activity performed in the routine life. The study predicted the progression towards metabolic syndrome independent of other potential confounding factors (standardized $\beta = -0.00085$, $P = 0.046$).

3. DIET

Insulin sensitivity is influenced not only by total energy intake but also by diet composition.

Intake of high monounsaturated fat diet greatly improves the insulin sensitivity when compared to a high-saturated-fat diet. But this beneficial effect is not seen in individuals whose total fat intake exceeds 38% of total energy **Vessby et al., 2000**.

Since dietary carbohydrate is a major precursor of blood glucose, increasing the amount of carbohydrate in the diet will raise blood glucose levels mainly in the postprandial period. Blood glucose concentration represents an important prompting factor for insulin release, hence a high carbohydrate diet will also lead to increased insulin levels. Whether the effects of a high-carbohydrate diet is more pronounced on glucose or insulin levels, depends on the insulin secretory capacity of the endocrine pancreas **Reaven, 1997**.

Glucose and lipid metabolism are strongly related and any imbalance of carbohydrate metabolism by a high-carbohydrate diet will also increase plasma triglycerides and decrease plasma HDL concentrations. Fibrinolysis is also deteriorated by a high-carbohydrate diet. This type of diet is also associated with an increase of PAI1 in blood **Lopez-Segura 1996**.

Other food constituents that affects the metabolic abnormalities and cardiovascular disease risk factors clustering in the metabolic syndrome are

- an excessive intake of alcohol (more than 30 gm/day) can raise both plasma triglyceride and blood pressure levels **Kiechl et al.,1996**.
- A high intake of sodium chloride can also increase the blood pressure.

4. AGEING

Ageing is defined as a sequence of morphological and functional changes which take place eventually. It also refers to the worsening of the biological functions after an individual has attained the maximum reproductive potential.

Many older individuals has increased abdominal fat despite a normal BMI, a factor that can decrease the utility of BMI as a predictor of Type 2 Diabetes Mellitus in older individuals. Ageing is also coupled with a raise in proinflammatory cytokines, which are known to hinder the insulin action. These cytokines are a resultant from both the age-associated accrual of visceral fat and secretion of proinflammatory cytokines by increasing number of senescent cells. Collectively these age-related alterations in metabolism and body fat distribution are active participants in a vicious cycle that can accelerate the ageing process and the onset of disease.

Another metabolic regulator derived from adipose tissue and linked to aging is the hormone adiponectin. Contrast to other fat-derived cytokines

that oppose insulin action, it is an insulin sensitizer with anti-inflammatory properties and a potent activator of AMP-activated protein kinase (AMPK). Adiponectin is paradoxically increased in lean individuals or in response to caloric restriction.

Ageing is associated with progressive loss in mitochondrial function in various tissues including skeletal muscle. Mitochondria are the major source of reactive oxygen species generation which causes oxidative damage to the macromolecules such as nuclear and mitochondrial DNA. This has led many to embrace the mitochondrial theory of ageing which states that mitochondrial dysfunction is a fundamental cause of cellular ageing and senescence.

The impaired mitochondrial function may impinge on insulin signalling is thought to involve reduced or incomplete β-oxidation of fatty acid substrates in metabolically active tissues such as liver and skeletal muscle. Investigations have reported an association between insulin resistance and impaired glucose tolerance with decreased mitochondrial oxidative activity and ATP synthesis in elderly and Type 2 Diabetic individuals. A study comparing mitochondrial function between healthy, lean, elderly individuals and matched lean, healthy young subjects found a

40% reduction in mitochondrial activity and oxidative phosphorylation in the elderly subjects with insulin resistance. Similarly, young insulin-resistant offspring of diabetic parents had reduced mitochondrial activity and increased fat content in skeletal muscle compared with insulin-sensitive control subjects.

The metabolic syndrome affects 44% of the U.S. population older than age 50 years. The age dependency of the syndrome's prevalence is seen in most populations around the world.

5. STRESS

Stress is defined as a state in which harmony or homeostasis is actually threatened or perceived to be so evoked by various cognitive (e.g., anxiety, fear, depression) and/or somatic stressors (e.g., pain, lipid accumulation, inflammation) when the hazard to homeostasis exceeds the threshold.

Psychosocial stress also plays a role in the pathogenesis of the metabolic syndrome. Continuous exposure to work-related stress can alter autonomic nervous system and neuroendocrine systems that control the reactions to stress. The neuroendocrine responses from the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system stimulate stress-related cortisol secretion. This contributes to the development of

chronic diseases such as dyslipidemia, abdominal obesity, hypertension and insulin resistance.

There are reports indicating that despite regular exercise and a proper diet, subjects under prolonged stress developed metabolic alterations including distinctive central obesity, biochemical changes and a slight hypertension towards the metabolic syndrome **Branth S et al., 2007**.There is a dose-response relation between exposure to work stressors and risk of the metabolic syndrome independent of other relevant risk factors **Chandola T et al., 2006.**

Although mechanisms behind this relationship remain unclear, prolonged exposure to work stress may affect the autonomic nervous system and neuroendocrine responses that can contribute to the development of the metabolic syndrome. Neuroendocrine responses to stress activate the HPA axis which results in the secretion of steroid hormones including corticosteroids and catecholamines which are major stress hormones. Cortisol is a corticosteroid hormone which is secreted from the adrenal cortex under the control of the HPA axis. During short-term acute stress, plasma cortisol levels are increased by the activation of the HPA axis for survival through homeostatic adjustments. However, frequent chronic stress may sustain elevated cortisol secretion that may damage to the HPA axis

with time resulting in a maladaptive process. Stress-related cortisol secretion has been linked with individual components of the metabolic syndrome. Hence, chronic stress at work is a major risk factor for the metabolic syndrome.

6. SMOKING

Smoking is widely accepted as a major risk factor for cardiovascular disease **Frati AC et al., 1996**. Previous studies have shown that smoking reduces insulin sensitivity, induces insulin resistance and enhances cardiovascular risk factors, such as elevated plasma triglycerides, decreased HDL–cholesterol and hyperglycemia.

In **Kawada's** 1-year follow-up study, current smokers had a higher risk of metabolic syndrome than nonsmokers, independent of age, body mass index, insulin resistance and other lifestyle factors. In contrast, ex-smokers did not have a significantly greater risk of metabolic syndrome than nonsmokers. The most effective way for smokers to reduce their risk of metabolic syndrome and cardiovascular disease is to stop smoking.

However, **Nakanishi et al., 2005**highlighted that smoking cessation is also associated with a 1.3-fold risk of metabolic syndrome as a result of subsequent body weight gain.

Mizuno et al., 2005 reported that waist circumference is significantly higher in obese subjects who smoke compared to the nonsmokers. Smoking seems to hasten the visceral fat accumulation and promote obesity-related disorders. Waist circumference is strongly associated with visceral fat mass which is influenced by the plasma cortisol concentration. Smokers have higher fasting plasma cortisol concentrations than nonsmokers. Higher cortisol concentrations may be a consequence of the stimulation of sympathetic nervous system activity which is induced by smoking **Williamson DF et al., 1991**.

Sex hormones may also get involved **Haarbo J et al., 1991**. In men, visceral fat increases when testosterone concentrations decrease and the administration of testosterone to middle-aged men reduces their visceral adipose tissue by increasing lipolysis. Smoking may reduce testosterone concentrations in men. Overall, these results suggest that in addition to excess cortisol and a reduction of testosterone in men may be involved in the effect of smoking on visceral adipose tissue hence leading to insulin resistance and development of metabolic syndrome.

7. DISRUPTED CHRONOBIOLOGY/SLEEP

Circadian rhythms are such an innate part of our lives. Recently, some studies have suggested that the disruption of the circadian system may be

causal for the manifestations of metabolic syndrome. Shift work, sleep deprivation and bright light exposure at night are related to increased adiposity and prevalence of metabolic syndrome. Surprisingly, circadian system impairment is not only the result of obligatory shift work schedules but is also an emerging issue in adolescent and young adults because their leisure activities result in voluntary sleep curtailment.

Epidemiological studies show that shift work is associated with obesity, hypertriglyceridemia, low HDL, abdominal obesity, diabetes and cardiovascular diseases.

Postprandial response are observed in shift workers with chrono-disruption of the melatonin profile. One of the most interesting recent findings is that shift work is an independent risk factor in the development of metabolic syndrome.

A study performed in day workers and shift workers indicated that shift workers had higher BMI even though the diet quality was even better in shift workers and the level of physical activity was similar between day and shift workers.

Interesting results come from the studies relating sleep duration and metabolic risk. The amount of sleep has declined by 1.5 hours over the past century with an important increase in obesity. Moreover, one-third of adults

sleep, less than 6 hours during night. Clinical studies show that healthy individuals restricted to 4 hours of sleep for six consecutive nights exhibit impaired glucose tolerance and reduced insulin responsiveness following a glucose challenge.

8. EXCESSIVE ALCOHOL CONSUMPTION

Protective and detrimental associations have been reported between alcohol consumption and the metabolic syndrome from the National Center for Chronic Disease Prevention and Health Promotion. This may be due to variations in drinking patterns and different alcohol effects on the metabolic syndrome components.

US Dietary Guidelines recommendations of not more than 1 drink per day for women or 2 drinks per day for men. Risk for the metabolic syndrome was increased by daily consumption that exceeded US Dietary Guidelines recommendations (odds ratio [OR], 1.60; 95% confidence interval [CI], 1.22 - 2.11) and by binge drinking once or more per week (OR, 1.51; 95% CI, 1.01 - 2.29). Drinking in excess of the Dietary Guidelines was associated with an increased risk for individual components of the metabolic syndrome.

The mechanisms underlying the relation of alcohol drinking with the metabolic syndrome may be explained by the relation with its components. Studies have reported that alcohol consumption is positively associated with

abdominal obesity. A meta-analysis of human experimental studies suggested that moderate alcohol drinking is positively associated with both HDL-cholesterol and triacylglycerol concentrations and the association did not differ significantly across beverage types. Consistent evidence on blood pressure elevated by heavy drinking has been reported while outcomes regarding light or moderate drinking have shown varied effects on blood pressure, such as beneficial, injurious, or similar effects compared with abstaining from alcohol. Observational studies have found an inverse association between alcohol consumption and glycemic control, atleast in part through enhanced insulin sensitivity. However, heavy liquor drinking was shown to increase the risk of Type 2 Diabetes Mellitus.

On the basis of these reports, it can be postulated that heavy drinking, in particular liquor drinking, has a detrimental influence on waist circumference, triacylglycerols, blood pressure and glucose leading to an increase in metabolic syndrome risk.

OCCUPATIONAL DRIVERS

Epidemiological studies have been done extensively to determine the prevalence and associated risk factors of the metabolic syndrome in the general population, yet less attention has been paid to its prevalence and risk factors for specific occupational groups such as in occupational drivers.

The reasons for excess risk of metabolic syndrome in occupational drivers are,

Lots of work and little pay

Drivers are expected to drive up to fourteen hours per day, receiving roughly ten hours off prior to the beginning of the next shift. Even though law regulating the amount of driving over the course of a day and week exist, these rules are usually bent and broken.

Drivers rarely receive more than one day of work off a week. The chance of death on the job is extremely high.

The work shift is also a risk factor for chronic diseases, like cardiovascular disease and metabolic disorders, because of altered circadian rhythm, lifestyle change and stress at work **Frost P et al., 2009.**

Unhealthy food habits

In addition to long hours, drivers rarely eat healthy food preparation. They often take high calorie diet. High carbohydrate meal consumption is related to hyperinsulinaemia, hyperglycaemia and hypertriglycemia that are well known to predict an increase of body fat in working individuals .The hours spent in sitting along with poor food choices make them obese.

Inaccessibility of Health care

The hours worked by drivers make it nearly impossible to make or keep medical appointments. This confines their options when a health problem arises leading many drivers to ignore symptoms.

Lack of regular exercise

Most drivers are poorly educated and they are not aware of the importance of doing regular exercises. They hardly find time to do regular exercise which in turn increases the chance of obesity.

Smoking & alcohol intake

In order to overcome the stress in their work place, drivers are prone to develop the habit of smoking and alcohol intake.

Road transport drivers are one of the professional groups whose activities have a strong impact on public safety. Moreover, study by **Evans L** on 1996 states that behavioural factors among professional drivers contribute considerably to the occurrence of traffic accidents.

Hence it is essential to initiate early detection of metabolic syndrome in this high risk population groups so that preventive measures can minimize the consequences. The aim of this study was to estimate the occurrence of metabolic syndrome among occupational drivers and studying the contributors of metabolic syndrome among them.

Evaluating the prevalence of the metabolic syndrome over time and identifying possible risk factors is critical to reduce the risk of cardiovascular disease and further reduce the premature death among this occupational cohort.

COMPLICATIONS

1. Cardiovascular Disease

The relative risk for cardiovascular disease in patients with the metabolic syndrome in the absence of diabetes is between 1.5-fold and 3-fold.

The pathophysiological mechanism by which the metabolic syndrome increases cardiovascular risk remains under debate. Earlier definitions by the World Health Organization and the European Group for the Study of Insulin Resistance emphasize the independent role of insulin resistance as the underlying component of the metabolic syndrome. Insulin resistance progresses toward hyperinsulinemia and hyperglycemia thus triggering peripheral vasoconstriction and sodium retention. Hepatic production of very low-density lipoprotein also increases, leading to hypertriglyceridemia, low HDL cholesterol, elevated apolipoprotein B, elevated small LDL cholesterol and consequently atherosclerosis. As a result of these lipid imbalances, individuals with the metabolic syndrome typically exhibit a prothrombotic and proinflammatory state.

More recent definitions by the NCEP, rNCEP and the International Diabetes Federation emphasize central obesity as the underlying component.

Adipocytes secrete mediators including TNF-α, leptin, adiponectin and resistin which lead to insulin resistance. In these definitions, it is postulated that central obesity causes systemic hypertension and dyslipidemia independently and through the induction of insulin resistance.

Regardless of which definition is used, insulin resistance and central obesity are postulated to be the key components of the metabolic syndrome and both lead to glucose intolerance and dysglycemia.

Cardiovascular disease is still most frequent cause of death among men under 65 years. In many developed countries it poses the largest public health problem. The etiology of cardiovascular disease is multifactorial .

Risk factors for Cardiovascular disease:

Modifiable	Non modifiable
Obesity Diabetes Hypertension Cigarette smoking Elevated serum cholesterol Stress Sedentary habits	Age Sex Genetic causes Family history Personality

Any individual can be categorized under the high risk group for developing atherosclerotic cardiovascular disorder, if any one of the above mentioned risk factor is present. 80% of deaths in patients with Type 2 Diabetes Mellitus were attributed by non-ischemic cardiovascular disease, stroke and myocardial infarction.

Independent of other risk factors, Type 2 Diabetes Mellitus not only increases the risk of cardiovascular morbidity and mortality, but also offers a synergistic interaction with other causative factors such as hypertension and dyslipidaemia.

Women are more vulnerable to the cardiovascular effects of Type 2 Diabetes Mellitus because they appear to lose the protective effects of estrogen in the premenopausal period **Willeit J et al., 1997**.

Rajeev Gupta et al., 2012 from Jaipur, conducted a study during the year 2002 to 2006 and stated that, the risk factors for developing cardiovascular disease in the urban population is always high.

Murthy et al., 2012 from Guntur district in Andhra Pradesh, high lightened that there is positive correlation between impaired glucose

tolerance and high levels of triglycerides and low density lipoprotein levels, which is linked with escalating prevalence of cardiovascular disease.

In a survey conducted at Tenali town, in Andhra Pradesh, between July 2009 and October 2009, the overall prevalence of cardiovascular disease was 5.4%. The prevalence rate of cardiovascular disease was11.3% in persons with diabetes, 23.5 % in those with impaired glucose tolerance and 3% in persons with normoglycemia **Dakshina Murthy et al., 2012.**

In a Finnish population diabetes augmented the risk of myocardial infarction fivefold and increased the risk of death from heart disease.

2. Type 2 Diabetes Mellitus

Overall, the risk for Type 2Diabetes Mellitus in patients with the metabolic syndrome is increased by 3- 5 fold. In addition to predicting cardiovascular disease morbidity and mortality, the metabolic syndrome is strongly associated with the development of Type 2 Diabetes Mellitus itself an important risk factor for cardiovascular disease . However, even before levels of blood glucose are high enough for a person to be diagnosed with

diabetes, raised blood glucose level and related changes in blood lipids (increase in triglycerides and decrease in the 'good' cholesterol HDL-c) increase a person's risk of cardiovascular disease.

Diabetes Mellitus is a multisystem disorder caused by the complex interaction of genetic and environmental factors. Diabetes is a metabolic disorder characterized by chronic hyperglycemia, resulting in various clinical manifestations. It is a chronic disease resulting in many dreaded complications like ocular, neurological, renal, cardiovascular and other intercurrent infections.

In the recent years, the age of onset of diabetes among Indians has been shifted to much younger age group. This will have a great impact in nation's economy and health. The prevalence of diabetes is more in urban population because of the epidemiological transition with decreased physical activity and unhealthy dietary patterns. When compared to other ethnic groups, there is high prevalence of stroke and cardiovascular disease among the Asian Indians. The incidence of micro vascular complications like diabetic retinopathy and neuropathy is low.

One of the main cause for increased incidence of premature cardiovascular disease and diabetes among Indians is due to genetic

factors and also the “Asian Indian Phenotype”. It includes central adiposity with increased waist circumference, insulin resistance, raised C-reactive protein levels, lower adiponectin and lower BMI. For prevention and postponement of onset of Diabetes Mellitus, early intervention through proper lifestyle modification and timely screening of individuals at risk should be carried out. It will result in great economic improvement of our country and reduces the burden of our society.

Clinical classification of Diabetes Mellitus:

1.Gestational Diabetes mellitus (GDM)

2. Impaired glucose tolerance (IGT)

3.Diabetes Mellitus (DM).

i) Type 1 DM -- IDDM -- Insulin dependent Diabetes Mellitus.

ii)Type 2 DM -- NIDDM--Non Insulin dependent Diabetes Mellitus.

iii) Secondary Diabetes --drug induced, genetic, hormonal, pancreatic and other abnormalities

iv)Malnutrition --related Diabetes Mellitus.

NIDDM is much more common than IDDM. The risk factors which determine the occurrence of Type 2 Diabetes Mellitus includes ethnicity, age and sex. The presence of other chronic disorders makes the condition more worse.

Currently the young adults and adolescents are in the high risk category for developing diabetes. Previously it was believed that Type 2 Diabetes Mellitus was a disease of elderly and middle aged people which has recently escalated in all age groups and is now seen also in younger age groups.

In children and young adults, there is an alarming increase in the prevalence of Type 2 Diabetes Mellitus. The National Health and Nutrition Examination Study (NHANES) data for 1999 to 2000 suggest that10.4% of 2-to 5-year olds, 15.3% of 6 to11 year olds,15.5% of 12 to 19 year olds have a BMI above 95^{th} percentile adjusted for sex and age. It represents an approximately 30% increase over the previously determined data for 1988 to 1994. Impaired glucose tolerance and Type 2 Diabetes Mellitus have now emerged as critical health issue in overweight children and young adults **Harris M I et al., 1998**.

The diagnosis of diabetes rests on the measurement of plasma glucose levels. The diagnostic criteria for diabetes were changed in 1997. The most significant changes were the level of fasting glucose that is recognized as diagnostic for diabetes which was decreased from**140 to 126mg/dl** and the introduction of a category of impaired fasting glucose.

Levels of hemoglobinA1c are not currently recommended for diagnosing diabetes. The major reasons are lack of standardization of assays for HbA1c, false positive and false negative results related to hemoglobinopathy and altered red cell survival and imperfect correlation between HbA1c and fasting and 2 hour plasma glucose.

However HbA1c remains the preferred method for monitoring the effectiveness of diabetes treatment **Kronenberg et al., 2008.**

CRITERIA FOR DIAGNOSIS OF DIABETES:

Test	Normoglycemia (mg/dl)	IFG (mg/dl)	IGT(mg/dl)	Diabetes Mellitus
FPG	< 100	100-125		≥126
2-hour PG	<140		140- 199	≥200
Casual plasma glucose concentration				≥200mg/dl plus symptoms of diabetes

Screening for Diabetes:

Undiagnosed diabetes is more common with an estimate lag of 5 to 7 years between the onset of diabetes and diagnosis. It is estimated that in upto 30% of affected people the disease are undiagnosed. Subjects with undiagnosed Type 2 Diabetes Mellitus and IGT are in a greater risk of developing peripheral vascular disease, stroke and coronary artery disease in their later life. Thus, this delay in the diagnosis of diabetes causes an increase in microvascular and macrovascular complications **Klein R et al., 1992**.

Impaired glucose tolerance or impaired fasting glucose sometimes referred to as "Pre-diabetes". The blood glucose levels in prediabetic

individuals are greater than normal. Various studies have recommended that the pre-diabetics should lose around 10% of their initial bod weight otherwise they are prone to develop diabetes in the next 10 years.

In 2007, data from American health department revealed that, around 57 million people are coming under the category of pre-diabetes. They also observed that one in 4 adults above the age of 20 years are having pre-diabetes.

According to **Chennai Urban Rural Epidemiology Study,** as per the WHO criteria, the prevalence of Impaired Glucose Tolerance was around 10% and that of diabetes was found to be around 15%.

In Chennai, there is an escalating prevalence of Diabetes Mellitus during the past 14 years **S Sandeep et al., 2011**.

Period of Study	**Increase in prevalence of DM(%)**
1989 – 1995	40%
1995- 2000	16%
2000- 2004	6%

3. Cerebrovascular accidents:

The presence of metabolic syndrome has been associated with an increased risk of stroke. In the National Health and Nutrition Examination Survey, the prevalence of metabolic syndrome was significantly higher in persons with a self-reported history of stroke (43.5%) than in subjects with no history of vascular disease (22.8%) **Ninomiya JK et al., 2004**. Metabolic syndrome was independently associated with stroke history in all ethnic groups and in both sexes (OR, 2.16; 95% CI, 1.48 to 3.16). The association between metabolic syndrome and stroke has been confirmed in elderly population and the frequency of metabolic syndrome has been reported to be significantly higher in patients with a history of atherothrombotic or nonembolic ischemic stroke **Milionis HJ et al., 2005.** This association helps the clinical use of the metabolic syndrome in the identification of subjects who are at an increased risk of experiencing a stroke.

Long-term follow-up population-based studies have demonstrated that healthy individuals with the metabolic syndrome are at a markedly increased risk for major cardiovascular events including stroke **McNeill AM et al., 2005**. In prospective studies, adjusted risk ratios for incident ischemic stroke associated with metabolic syndrome range from2.1 to 2.47 and a hazard ratio as high as 5.15 has been reported. This predictive capacity appears not to be influenced by the metabolic syndrome definition used and shows no significant variation across the sex, age or ethnic groups. The risk for incident ischemic stroke seems to increase with the increasing number of components of the metabolic syndrome, all of which are individually associated with an increased risk for future cerebral ischemic events **Chen HJ et al., 2006.**

4. Nonalcoholic Fatty Liver Disease(NAFLD):

Fatty liver is relatively common. Non- alcoholic fatty liver disease is often associated with features of the metabolic syndrome carrying an increased risk for the development of non-alcoholic steatohepatitis (NASH), the inflammatory form of liver steatosis. Data from epidemiological study confirm that obesity, diabetes, hypertension and dyslipidemia are frequently found in NAFLD and worsen its prognosis because of increased risk of

fibrotic evolution eventually leading to liver cirrhosis. As the prevalence of overweight/obesity and the metabolic syndrome increases, NASH may become one of the more common causes of end-stage liver disease and hepatocellular carcinoma.

5.Chronic kidney disease

People with metabolic syndrome have a 55% increased risk of developing renal disorders especially lower kidney function, indicative of kidney disease. Kidney disease risk increases as the number of metabolic syndrome components increases.

Hoehner et al., 2002 correlated the metabolic syndrome profile and microalbuminuria in a crosssectional study of American Indians from **Wisconsin and Minnesota**. Individuals with three or more metabolic syndrome traits had a 2.3-fold increased odds of having microalbuminuria compared with a control group without the syndrome.

Palaniappanet al., 2004 found a statistical association between metabolic syndrome and microalbuminuria. Also discovered a significant correlation between number of metabolic syndrome factors and GFR.

Individual traits that confer greatest risk were hypertension and hyperglycemia.

6.Polycystic Ovary Syndrome (PCOS)

Many patients with PCOS also have features of the metabolic syndrome which includes insulin resistance, obesity and dyslipidemia, suggestive of an increased risk for cardiovascular disease. Increased awareness of this overlap advocates therapies that improve insulin resistance and often ameliorate PCOS symptoms. This is one condition commonly detected in a younger age group and associated with a high risk of progression to diabetes.

PCOS is highly associated with the metabolic syndrome with a prevalence between 40 and 50%. Women with PCOS are 2–4 times more likely to have the metabolic syndrome than are women without PCOS.

Unlike the metabolic syndrome with its largely asymptomatic risk factors, PCOS occurs with overt symptoms like infertility, hirsutism and acne. Eventhough these are the problems that bring women to the attention of health care providers, their presence affords the opportunity to intervene

early with counselling and if needed, medications to modify the risk profile for later development of the metabolic syndrome or cardiovascular disease.

7. Obstructive Sleep Apnoea

The combination of metabolic syndrome and obstructive sleep apnoea has been termed **"Syndrome Z".** The prevalence of both obstructive sleep apnoea and metabolic syndrome are increasing worldwide which is linked to the epidemic of obesity. Apart from their epidemiologic relationship, growing evidence suggests that obstructive sleep apnoea may be related to metabolic syndrome.

Obstructive sleep apnoea is associated with alterations in the HPA axis that may promote metabolic syndrome. The hypoxia associated with obstructive sleep apnoea seems to trigger an oxidative stress that may promote development of metabolic syndrome. Several inflammatory markers and mediators including CRP, TNF-α and IL-6 are elevated in patients with obstructive sleep apnoea and may play a role in the development of metabolic syndrome. The adipokines like leptin, ghrelin and adiponectin are dys-regulated in patients with obstructive sleep apnoea in ways that promote a positive energy balance, obesity and metabolic syndrome. Sleep deprivation from obstructive sleep apnoea may also be

implicated due to its effects on both metabolic regulation and systemic inflammation. Acute sleep deprivation is associated with altered glucose homeostasis resulting in high blood glucose levels, insulin resistance and risk of diabetes. Identification of mechanistic interactions between obstructive sleep apnoea and metabolic syndrome may suggest opportunities by which the pathophysiology of obstructive sleep apnoea can be interrupted to prevent manifestations of the metabolic syndrome.

TREATMENT:

The National Cholesterol Education Programme (NCEP) Adult Treatment Panel (ATP) III defined the criteria for the diagnosis of the metabolic syndrome and established the basic principles for its management. According to this, treatment involves correcting the underlying insulin resistance through lifestyle modification (eg, weight reduction and increased physical activity) and possibly by drugs. The coexistent risk factors (mainly dyslipidemia and hypertension) should also be addressed.

Goals of Treatment

The major goal of treating metabolic syndrome is to reduce the risk of cardiovascular disease. Treatment is directed first at lowering LDL cholesterol and high blood pressure and managing diabetes (if these conditions are present).

The second goal of treatment is to prevent the onset of Type 2 Diabetes Mellitus (if it hasn't already developed). If diabetes is present, the goal of treatment is to reduce your risk of the long-term complications which includes heart and renal disease, vision loss and foot or leg amputation.

Healthy lifestyle changes are the first line of treatment for metabolic syndrome. Lifestyle changes include losing weight, being physically active, taking a healthy diet and to quit smoking.

Lifestyle Changes

Losing Weight

The long-range target is to lower the body mass index (BMI) to < 25. With weight reduction, the improvement in insulin sensitivity is often accompanied by favourable modifications in many components of the metabolic syndrome. In general, recommendations for weight reduction include a combination of caloric restriction in diet, increased physical activity and behaviour modification.

Losing visceral fat

The following recommendations have been recognized as being helpful in reducing levels of visceral fat.

- Regular exercise
- Healthy, balanced diet
- Good night's sleep

- Reducing stress level
- Limiting alcohol intake
- Quitting smoking

Harvard University states that diet and exercise are found to be more effective at reducing visceral fat than the fat around the hips and thighs. When people slim down through exercise and diet, visceral fat reduces twice as fast as subcutaneous fat.

A research study, published in 2011 by **Newcastle University**, reported that a very low calorie diet can significantly reduce levels of visceral fat in people with Type 2 Diabetes Mellitus. Along with the reduction in visceral fat, the study participants showed improved blood glucose levels and a number of them were able to reduce or come off their diabetes medication.

Following a Healthy Diet

A heart healthy diet is an important part of a healthy lifestyle

A healthy diet includes

1. A variety of vegetables and fruits. A good rule is to try to fill half of your plate with vegetables and fruits.
2. A healthy diet also includes whole grains, low-fat dairy products and protein foods, such as lean meats, seafood, soy products nuts, seeds, beans and peas.
3. Choosing and preparing foods with little sodium (salt). Too much of salt can raise the risk for high blood pressure.
4. Avoiding foods and drinks that are high in added sugars. For example, taking water instead of sugary drinks like soda.
5. Avoiding high consumption of alcohol since it adds extra calories, which can cause weight gain.

Studies comparing ethnically similar populations exposed to different dietary environments suggested that Westernized diets are strongly associated with a higher risk of developing metabolic syndrome. On the other hand, diets rich in dairy, fish and cereals may be associated with a lower risk of developing metabolic syndrome **Ruidavets JB et al., 2007**.

Being Physically Active

Exercise is thought to be an important intervention **Roberts CK et al., 2013**.The current recommendation for patients is to perform regular

moderate-intensity physical activity for at least 30 minutes continuously at least 5 days per week (ideally, 7 days per week). However maintaining long-term adherence remains a challenge **Fappa E et al., 2007**.A study by **Bateman et al., 2011** concluded that aerobic training is the most efficient mode of exercise for improving the health.

In one prospective study, cardiorespiratory fitness was linked to the risk of developing metabolic syndrome in a dose-dependent manner with patients in the highest category of fitness having the lowest risk of developing new-onset metabolic syndrome **Shuval K et al., 2012**.

Evidence suggests that excessive sitting and other behaviours that are low in activity and energy expenditure may trigger unique cellular responses that contribute to the development of metabolic syndrome.

Smoking

Quit smoking. Smoking can raise the risk for heart disease and heart attack. Smoking cessation can be achieved with or without assistance from healthcare providers or the use of medications. However, a combination of personal efforts and medications proves more effective to many smokers. Methods that are found to be effective include interventions directed at or

via health care providers and health care systems; medications including nicotine replacement therapy (NRT), individual and group counselling.

Several hospitals, workplaces and community groups offer classes to help people quit smoking.

MEDICATIONS:

If lifestyle changes are not enough, medicines are used to treat and control risk factors such as high blood pressure, high triglycerides, low HDL cholesterol and high blood sugar.

LDL Cholesterol

According to NCEP:ATPIII, for patients with the metabolic syndrome and diabetes, LDL cholesterol should be reduced to <100 mg/dl and perhaps further in patients with a history of cardiovascular disease.

Diets restricted in saturated fats (<7% of calories), trans-fats (as few as possible), and cholesterol (<200 mg daily) should be applied aggressively. If LDL cholesterol remains above goal, pharmacologic intervention is needed.

Statins (HMG-CoA reductase inhibitors) which produce a 20–60% lowering of LDL cholesterol are usually the first choice for medication intervention. Fibrates are best employed to lower LDL cholesterol when both LDL cholesterol and triglycerides are elevated.

Triglycerides

A fasting triglyceride value of <150 mg/dl is recommended. Generally, the response of fasting triglycerides relates to the amount of weight reduction achieved. A weight reduction of >10% is necessary to lower fasting triglycerides.

A fibrate (gemfibrozil or fenofibrate) is the drug of choice to lower fasting triglycerides and typically achieve a 35–50% reduction. Other drugs that lower triglycerides include statins, nicotinic acid and high doses of omega-3 fatty acids.

HDL Cholesterol

Beyond weight reduction, there are very few lipid-modifying compounds that increase HDL cholesterol. Nicotinic acid is the only currently available drug with predictable HDL cholesterol-raising properties. The response is dose-related and can raise HDL cholesterol ~30% above

baseline. There is limited evidence at present that raising HDL has a benefit on cardiovascular events independent of lowering LDL cholesterol mainly in patients with the metabolic syndrome.

Blood Pressure

Treatment of hypertension had been based on the recommendations of the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC-7) guidelines, to attain a goal, blood pressure of less than 140/90 mm Hg or, less than 130/80 mm Hg in patients meeting diagnostic criteria for Diabetes Mellitus.

In patients with the metabolic syndrome without diabetes, the first choice of antihypertensive should usually be an angiotensin-converting enzyme (ACE) inhibitor or an angiotensin II receptor blocker, because these two classes of drugs reduce the incidence of new-onset Type 2 Diabetes Mellitus. In all hypertensive patients, a sodium-restricted diet enriched with fruits and vegetables and low-fat dairy products should be advised. Home monitoring of blood pressure may help in maintaining good blood pressure control.

Impaired Fasting Glucose (IFG)

In patients with the metabolic syndrome and Type 2 Diabetes Mellitus aggressive glycemic control will favourably modify fasting triglycerides and/or HDL cholesterol. In patients with IFG without a diagnosis of diabetes, a lifestyle modification that includes weight reduction, dietary caloric restriction and increased physical activity has been shown to reduce the incidence of Type 2 Diabetes Mellitus. Metformin has also been shown to decrease the incidence of diabetes but the effect was less than that seen with lifestyle intervention.

Insulin Resistance

An insulin-sensitizing agent such as metformin is typically used as a first drug in the treatment of patients with metabolic syndrome. Few literature suggests that metformin help to reverse the pathophysiological changes of metabolic syndrome. Several drugs such as biguanides, thiazolidinediones increase insulin sensitivity. Both metformin and thiazolidinediones enhance insulin action in the liver and suppress endogenous glucose synthesis. Thiazolidinediones but not metformin has been found to improve insulin-mediated glucose uptake in muscle and adipose tissue. Both the drugs also have beneficial effects in patients with

NAFLD and PCOS and the drugs have been shown to reduce markers of inflammation and small dense LDL.

Cardiovascular complications

Aspirin may contribute to the primary prevention of cardiovascular complications in metabolic syndrome mainly in patients with at least an intermediate risk of suffering a cardiovascular event (ie, >6% 10-y risk). Low-dose aspirin can help reduce the risk of blood clots particularly for people in whom risk of heart disease is high **Blaha MJ et al., 2008**.

Surgical considerations

At present no surgical interventions for metabolic syndrome have been widely accepted. Yet, trials of bariatric surgery in patients who were morbidly obese and had metabolic syndrome showed beneficial results including decrease in insulin resistance and lower levels of inflammatory cytokines.

Visceral fat around abdominal organs cannot be liposectioned. Liposuction surgery removes only the subcutaneous fat. Risk factors including high blood pressure, hypercholesterolemia and insulin resistance are not improved with loss of subcutaneous fat.

Importantly, metabolic syndrome raises specific perioperative issues that should be considered in patients with metabolic syndrome undergoing any major surgical procedure **Bagry HS et al., 2008**.

Treatment of obstructive sleep apnoea syndrome

Treatment of associated obstructive sleep apnoea may play a significant role in the management of metabolic syndrome **Drager LF et al., 2013**. In a 2011 study, patients with moderate obstructive sleep apnoea who used continuous positive airway pressure (CPAP) therapy for 3 months showed significant improvements in their metabolic profile including reduction in blood pressure, LDL-cholesterol, triglycerides and glycated hemoglobin levels. Moreover, reversal of metabolic syndrome occurred to a greater degree in the CPAP therapy group than in patients who underwent treatment (13% vs 1%, respectively) **Sharma SK et al., 2011**.

MATERIALS AND METHODS

Design of the study:

It is a cross sectional study.

Place of study:

The study was conducted in Jeyavilas bus depot, Madurai.

Collaborating Department :

Department of Biochemistry, Madurai Medical College, Madurai.

Study subjects:

A total of 100 subjects in the age group of 25 to 60 years were selected from Jeyavilas bus depot, Madurai.

Inclusion criteria:

Men who were drivers by occupation for > 5 years with minimum 8 hours of driving per day.

Age group 25-60 years.

Exclusion criteria:

Females.

Endocrine disorders like – Hypothyroidism, Cushing's disease.

Chronic renal disease.

On medication – Steroid, Beta blokers, Thiazides etc.

Ascites.

Materials used for study:

1.Proforma – to record the anthropometric measurements of the subjects and the clinical findings.

2.Perceived stress scale 10 (PSS 10) questionnaire in Tamil – to assess the stress level.

3.Portable weighing machine – to record the body weight in kilograms.

4.Stadiometer – to measure the standing height in centimeters.

5.Non elastic inch tape - to measure Waist circumference and Hip circumference in centimeters.

6. Standardized mercury sphygmomanometer – to record the Blood Pressure in mm of Hg.

7. EM 360 Fully Automated analyzer - for estimating plasma glucose and lipid profile.

METHODOLOGY:

The study was initiated with the approval of Institutional ethical committee, Madurai Medical College, Madurai. The study was carried out after explaining the procedures in detail and getting written informed consent from the subjects.

The experimental protocol includes,

1) Recording of a detailed history including family history of Cardiovascular disease, Hypertension, Diabetes Mellitus, history of smoking, alcohol consumption, physical activity and number of years spent in driving as an occupation.
2) Perceived stress level was assessed using a standard questionnaire-Perceived stress scale 10 (PSS 10). Subjects were asked to fill up the PSS 10 questionnaire.
3) Measurement of Anthropometric Indices:

   The subjects were asked to stand erect, with their arms relaxed at their side and with feet together.

Weight (in kilograms) was recorded using a portable standard weighing machine. Weight was measured to the nearest 0.5 kg in subjects wearing inner clothing and without shoes after they had emptied their pockets.

Height (in centimeters) was measured to the nearest 0.5 cm by asking the patient to stand erect without shoes and the vertical height was measured using a stadiometer.

Body Mass Index (BMI) was computed as the weight in kilograms divided by the square of the height in meters using Quetelet's Index.

BMI = Weight (Kg)/ Height (m^2).

Waist circumference was measured to the nearest 0.5 cm with an inch tape at the point midpoint between the lower rib margin and the iliac crest at the end of normal expiration with the subject in standing position.

According to the National Cholesterol Education Programme Adult Treatment Panel III (2001)Waist circumference ≥ 90 cm in males, ≥ 80 cm in females is considered as central obesity.

1) Measurement of Blood pressure:

Documentation of blood pressure was done using the standard sphygmomanometer with cuff size 25×12.5cms. Before taking measurement the subject was seated quietly for 15 minutes in a quiet room with a comfortable room temperature. For final analysis, the mean value of the two blood pressure recordings obtained at 5 minutes interval from the left arm of the subjects in the sitting posture is taken.

2) Blood investigations:

After an overnight fasting for 8-10 hours blood sample was collected.

The following investigations were done :

For venous blood collection antecubital vein of front of forearm was selected. Skin is sterilized over the vein with a spirit cotton swab. A disposable sterile needle fixed to a disposable syringe of 10ml capacity and the desired amount of blood was collected.

1. Fasting Plasma Glucose.

For separation of serum, blood taken into a plain vial is first allowed to clot and then centrifuged at 3000 rpm for 5 minutes.

This separated serum was used to estimate:

2.HDL cholesterol

3.Triglycerides

COLLECTION OF BLOOD SAMPLE

Fasting plasma sugar was estimated by glucose oxidase peroxidase (GOD-POD) method.

Serum HDL - cholesterol and Triglycerides were measured by direct enzymatic method using an auto-analyser (ERBA-XL-300).

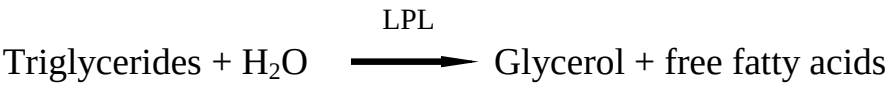
ESTIMATION OF TRIGLYCERIDES:

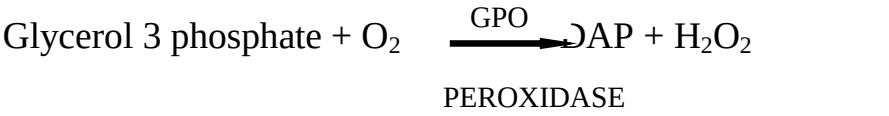
METHOD: GPO-PAP method, endpoint

METHODOLOGY:

Colorimetric, enzymatic method with glycerol phosphate oxidase.

PRINCIPLE:





LPL- Lipoprotein lipase

GK- Glycerol kinase

GPO- Glycerol Phosphate Oxidase

DAP-Dihydroxy Acetone Phosphate

ATP- Adenosine Tri Phosphate

4AAP- 4Amino Anti Pyrine

DHBS-3,5Dichloro-2Hydroxy Benzene Sulfonate

Lipoprotein lipase catalyzed hydrolysis of triacylglycerol yield glycerol which is phosphorylated by glycerol kinase using ATP to glycerol-3-phosphate which upon oxidation yields dihydroxy acetone phosphate and hydrogen peroxide. The hydrogen peroxide reacts with phenolic compound and 4amino antipyrine to form a coloured complex.

The intensity of Quinoneimine dye formed is proportional to the triglyceride concentration in the sample when measured at 505 nm (500-540nm).

REAGENT:

Reagent 1 (Enzymes/chromogen)

Reagent 2 (Buffer)

Triglycerides standard concentration- 200mg/dl

REAGENT PREPARATION:

The working reagent was prepared by mixing 4 parts of R1 with 1 part of R2. Stable for 90 days at 2-8 ˚C.

Sample: Unhemolysed serum collected after 12 hours of fasting.

ASSAY PROCEDURE:

Pipette into tubes marked	**Blank**	**Standard**	**Test**
Working reagent	1000µl	1000µl	1000µl
Distilled water	10µl	-	-
Standard	-	10µl	-
Sample	-	-	10µl

Mixed and incubated for 10minutesat room temperature. Absorbance were read at 505nm for standard and sample against reagent blank.

CALCULATION:

$$\text{Triglycerides(mg/dl)} = \frac{\text{Absorbance of test}}{\text{Absorbance of standard}} \times \text{Concentration of standard}$$

REFERENCE VALUES:

Serum/plasma	37˚C
Normal fasting level	25-160mg/dl

Linearity – upto 1000mg/dl

Sensitivity- 2mg/dl

ESTIMATION OF HDL CHOLESTEROL:

METHOD: Phosphotungstic acid method, endpoint

PRINCIPLE:

Chylomicrons(CM), LDL and VLDL are precipitated from serum or plasma with phosphotungstate in the presence of divalent cations such as Magnesium. The HDL cholesterol remains unaffected in the supernatant and is estimated using cholesterol reagent.

$$\text{Serum/plasma} \xrightarrow{\text{Phosphotungstic acid, } Mg^{2+}} \underset{\text{(supernatant)}}{\text{HDL}} + \underset{\text{(precipitate)}}{\text{(LDL+VLDL+CM)}}$$

REAGENT COMPOSITION:

Reagent1: precipitating reagent

Phosphotungstic acid	2.4mmol/l
Magnesium chloride	40mmol/l

HDL cholesterol standard – 25mg/dl

SAMPLE: Unhemolysed serum used

PRECIPITATION:

Precipitation of LDL,VLDL and Chylomicrons done as follows:

Pipette	Volume
Sample	250µl
Precipitating reagent	500µl

Mixed well and the reaction mixture was allowed to stand for 10 minutes at room temperature centrifuged at 4000 rpm for 10minutes and obtain a clear supernatant solution. The supernatant was used to determine the concentration of HDL cholesterol in the sample.

ASSAY PROCEDURE:

Pipette into tubes marked	Blank	Standard	Test
Cholesterol working reagent	1000µl	1000µl	1000µl
Distilled water	50µl	-	-
HDL standard	-	50µl	-
Supernatant	-	-	50µl

Mixed well and incubated for 10 minutes at room temperature.

The absorbance of the standard and the test samples were read at 505 nm against reagent blank.

CALCULATION

$$\text{HDL cholesterol (mg/dl)} = \frac{\text{Absorbance of test}}{\text{Absorbance of standard}} \times \text{conc.of standard} \times \text{dilutionfactor}$$

$$= \frac{\text{Absorbance of the test}}{\text{Absorbance of the standard}} \times 25 \times 3$$

$$= \frac{\text{Absorbance of the test}}{\text{Absorbance of the standard}} \times 75$$

Linearity- upto 125mg/dl.

ESTIMATION OF GLUCOSE:

METHOD: Glucose oxidase- peroxidase method, end point/fixed time.

PRINCIPLE: Glucose in the sample is oxidized to yield gluconic acid and hydrogen peroxide in the presence of glucose oxidase. The enzyme peroxidase catalyses oxidative coupling of 4 –amino antipyrine with phenol to yield a coloured quinoneimine complex with absorbance proportional to the concentration of glucose in the sample.

Glucose oxidase

Glucose + O_2 + H_2O ⟶ Gluconicacid + Hydrogen peroxide

Peroxidase

H_2O_2 + Phenol+4 AAP ⟶ Quinoneimine dye + 2 H_2O_2

Glucose standard : 100mg/dl

Specimen: Fresh unhemolysed serum used.

ASSAY PROCEDURE:

Enzyme reagent	1ml	1ml	1ml
Blank	10µl	-	
Standard	-	10µl	-
Test	-	-	10µl

Mixed well after each addition and incubated at 37˚C for 5 minutes. The absorbance of the standard and the test were read against reagent blank at 505nm.

CALCULATION:

$$\text{Glucose (mg/dl)} = \frac{\text{Absorbance of test}}{\text{Absorbance of standard}} \times \text{concentration of standard (100mg/dl)}$$

Linearity upto 500mg/dl by endpoint method.

NORMAL VALUES:

- Glucose fasting= 70-110 mg/dl
- Glucose postprandial= 90-140 mg/dl.

RESULTS AND OBSERVATION

STATISTICAL ANALYSIS

The association between the risk factors and the outcome variables was analysed using Pearson's Chi Square test. The statistical analysis was done using **SPSS (Statistical Package for Social Sciences) software version 21.** The **statistical significance** was drawn at **'p' value < 0.05.** 'p' value **<0.05** was considered as **statistically significant.**

Table-1: Association of influence of BMI with the occurrence of metabolic syndrome among occupational drivers

S.No	BMI (kg/m^2)	Presence of MetS		'p' value
		No	Yes	
1	< 18.5	32	1	< 0.001
2	18.5-24.9	19	4	
3	25-29.9	14	13	
4	≥ 30	2	15	

When comparing the occurrence of metabolic syndrome with BMI, there was a significant increase in the occurrence of metabolic syndrome as the BMI increases.

Results analysed using Pearson's Chi Square test revealed a statistically **very significant 'p' value (p <0.001).**

Table-2: Association of influence of stress with the occurrence of metabolic syndrome among occupational drivers

S.No	PSS 10 score	Presence of MetS		'p' value
		No	Yes	
1	≤ 12	26	8	0.001
2	13-23	33	10	
3	24-29	8	15	

When comparing the occurrence of metabolic syndrome with stress level using Perceived Stress Scale (PSS 10), there was a significant increase in the occurrence of metabolic syndrome as the stress level increases.

Results analysed using Pearson's Chi Square test revealed a statistically **very significant 'p' value (p 0.001).**

Table-3: Association of influence of smoking with the occurrence of metabolic syndrome among occupational drivers

S.No	History of smoking	Presence of MetS		'p' value
		No	Yes	
1	No	46	13	0.005
2	Yes	21	20	

When comparing the occurrence of metabolic syndrome with smoking, there was a significant increase in the occurrence of metabolic syndrome in smokers than in non smokers.

Results analysed using Pearson's Chi Square test revealed a statistically **significant 'p' value (p 0.005).**

Table-4: Association of influence of alcohol consumption with the occurrence of metabolic syndrome among occupational drivers

S.No	History of alcohol consumption	Presence of MetS		'p' value
		No	Yes	
1	No	41	15	0.136
2	Yes	26	18	

When comparing the occurrence of metabolic syndrome with alcohol intake, there was no significant difference in the occurrence of metabolic syndrome in person who take alcohol and those who do not take alcohol.

Results analysed using Pearson's Chi Square test revealed a statistically **insignificant 'p' value (p 0.136).**

Table-5: Association of influence of duration of service with the occurrence of metabolic syndrome among occupational drivers

S.No	Duration of service (yrs)	Presence of MetS		'p' value
		No	Yes	
1	5-9	29	4	<0.001
2	10-19	20	6	
3	20-29	13	10	
4	≥30	5	13	

When comparing the occurrence of metabolic syndrome with duration of service, there was a significant increase in the occurrence of metabolic syndrome as the duration of service increases.

Results analysed using Pearson's Chi Square test revealed a statistically **very significant 'p' value (p <0.001).**

DISCUSSION

In the past few years growing attention has been paid to the metabolic syndrome since early reorganization can prevent from its complications. However to date, data on its occurrence and the underlying risk factors are very limited among occupational drivers. Therefore the purpose of this study is to evaluate the occurrence of metabolic syndrome and to assess its risk factors among this special occupational group.

According to **Moreno et al., 2004** this category of workers in Brazil has a higher chance of obesity, physical inactivity, unhealthy diet, smoking habits, high levels of cholesterol, hypertension and obstructive sleep apnoea in comparison to the general population in Brazil.

In India very few studies had been done to find the prevalence of metabolic syndrome and most of the studies which are available in literature had used ATP-III. But **Deepa et al.,2006** compared the prevalence of metabolic syndrome in South Indian population by various definitions and found that by IDF 25.8% individuals more than 20 years of age were having metabolic syndrome as compared to 18.3% by ATP-III.

Higher prevalence by using IDF criteria can be explained by the lower cut-off points adopted by this new definition. In IDF criteria,

central obesity is the major criterion, its cut –off is ethnicity specific and is lower for Indians than used by original ATP-III. Another difference between two definitions is lower cut off for fasting blood sugar by IDF, which is >100 mg/dl as compared to >110 mg/dl in ATP-III. The higher prevalence by new IDF definitions is comparable with other reports.

The present study shows the occurrence of metabolic syndrome to about 33% of the study population, which reveals higher prevalence of metabolic syndrome than that of the general population. In a cross sectional study, among drivers in the central part of Iran, 35.9% of the participants suffered metabolic syndrome based on ATP III criteria **Saberi HR et al., 2011**. In a comparative study done in Norway on cardiovascular risk factors among bus drivers and industrial workers, the results showed that bus drivers had higher mean values of serum cholesterol level, serum triglycerides level, systolic and diastolic blood pressure.

Smoking and obesity are the main causes of preventable morbidity and mortality in developed countries **Ezzati Met al., 2004.**

Obesity is due to combination of inadequate exercise, high caloric diet, inadequate physical activity and genetic predisposition.

In developing countries like India, obesity affects most of the adults and children resulting in many public health problems. The prevalence of obesity is around 15% of children in U.S. 20% of children in Australia and 14% of children in Egypt.

The reason why bus driver showed high BMI (BMI $\geq$ 25 kg/m^2) and waist circumference (WC $\geq$ 90cm) is due to their irregular eating habits and low physical activity rates due to sitting on the job. As abdominal visceral adipose tissue increases, blood glucose and TG increases, HDL decreases and hypertension or diabetes increases **Liu JK et al., 2010.**

The present study reveals that occupational drivers are more prone for obesity. This category is more in drivers with metabolic syndrome. High BMI contributed significantly towards the development of metabolic syndrome. The study carried out by **Shakhatreh et al., 2000** in Saudi Arabia concluded that 73.2% of the drivers were obese. In Taiwan, obesity was seen in 9.6% of 2297 bus drivers while 4.6% for the workers. In Poland, obesity in drivers was 17.4%. In another study conducted in Mexico on professional drivers, it was found that the prevalence of obesity and overweight was 22.5 and 52.7%, respectively, which was higher than the general population of Mexico.

A study carried out in the United States involving more than 600 thousand workers found the highest prevalence of obesity to be among male employees who work in highway transportation services (31.7%). A study involving a representative sample of the Australian in productive age compared ten different functional categories with regard to the risk of obesity and found that male employees of the transportation industry had a higher risk of overweight and obesity **Aguilar-Zinser JV et al., 2007**.

Perceived stress, which measures the degree to which a person appraises situations in his or her life as stressful is one of several psychosocial factors related to work-related stress **Cohen S et al., 1983**. Perceived work stress has been linked with CVD as well as components of the metabolic syndrome **Rosmond R et al., 2000**. Chronic stress is associated with an increased risk of the metabolic syndrome and can be an underlying cause of the metabolic syndrome by dysregulating the HPA axis **Gohil B et al., 2001**.

Hills and Norvell suggested that the PSS is a significant and important predictor of stress-induced consequences including exhaustion, physical symptoms and job dissatisfaction in highway patrol officers. **Franke et al., 2002** found that high PSS was associated with cardiovascular disease risk. In contrast, however, **Yoo and colleagues** found that the PSS

was not significantly associated with the metabolic syndrome (r = 0.047).Therefore, chronic stress at work is an important risk factor for the metabolic syndrome.

Franke and colleagues found that higher levels of perceived stress are directly associated with increased cardiovascular disease prevalence, in addition to increased prevalence of dyslipidemia , hypertension and physical inactivity. Recent studies also suggest that stress at work may influence the pathogenesis of the metabolic syndrome.

Chandola and coworkers conducted a prospective cohort study with an average of 14 years of follow-up on 10,308 men and women. This study found that employees with chronic work stress are more than twice as likely to have the metabolic syndrome than those without stress at work, after controlling for age and employment grade (odd ratio [OR] = 2.25, 95% CI = 1.31 – 3.85).

Working environment of drivers may also play a role in developing metabolic syndrome.

In the present study, analysis revealed smoking to contribute significantly to the development of metabolic syndrome. Compared with nonsmokers, active smokers usually have more severe insulin resistance and

hyperinsulinemia which can increase the risk of Type 2 Diabetes Mellitus **He Y et al., 2009**.

Several studies have also shown that smoking is associated with metabolic abnormalities and increases the risk of metabolic syndrome **Nakanishi et al., 2005**. This study reported that subjects who habitually smoked tobacco had a 1.07–1.66 fold greater risk of developing metabolic syndrome than subjects who did not smoke. **Weitzman et al., 2005** have demonstrated the relationship between tobacco smoke and the severity of metabolic syndrome. The authors reported that exposure to tobacco smoke, either active or passive smoking is associated with a fourfold increase in the risk of the metabolic syndrome among adolescents who are overweight or at risk of overweight. **Saarni et al., 2009** investigated the association between adolescent smoking and overweight or abdominal obesity in adulthood. The result is that smoking is a risk factor for abdominal obesity in both sexes and for overweight in women.

Several earlier small-scale studies have reported smoking to be associated with higher prevalence of metabolic syndrome. The positive dose–response relationship between the amount of tobacco smoked and the prevalence of metabolic syndrome that we observed is also consistent with previous studies **Weitzman M et al., 2005**.

Some earlier epidemiologic studies relating alcohol consumption to the metabolic syndrome have lent data supporting that a minimal amount of drinking has protective effects on the prevalence of the metabolic syndrome compared with nondrinking **Freiberg MS et al., 2004**. By contrast, **Fan et al., 2006** examined lifetime drinking pattern and found that the prevalence of the metabolic syndrome was directly increased with lifetime drinking intensity (total drinks/drinking days over lifetime).It has been suggested that 5 to 30 gm/day of alcohol consumption may be favourable for Type 2 Diabetes Mellitus. Observational studies have found an inverse association between alcohol consumption and glycemic control at least in part through enhanced insulin sensitivity **Meyer KA et al., 2003**.

The study also observed that the metabolic syndrome components contributing to the increase are hypertension and an elevated concentrations of fasting glucose and triacylglycerols for both men and women and central obesity for women. Thus, suggesting that average moderate drinking over the lifetime does not have favourable effects on the metabolic syndrome.

Because of the nature of their work, they are used to the habit of smoking and alcoholism. In the present study it is found that with an increase in the duration of service, there is an increase in the occurrence of

metabolic syndrome. This indicates the combined role of stress, sedentary life style and smoking with the occurrence of metabolic syndrome.

CONCLUSION

Drivers sit for long hours and walk less compared to the general public. In addition, physical activity involvement and physical demands during the driving are usually limited and insufficient to maintain physical fitness.

Studies in recent decades have demonstrated that workers in the transportation industry are at greater risk of an incorrect diet and sedentary behavior **Bigert C et al., 2003**.

Our study showed influences of obesity, stress, smoking and alcohol consumption on the occurrence of metabolic syndrome in occupational drivers.

The most effective measures to improve insulin sensitivity in metabolic syndrome affected individuals are exercise and weight loss. Both modalities are effective and can be additive in their ability to improve insulin action.

"Regular exercise" is required for healthy aging!!

Hence a motivation a minimum of three exercise training sessions a week to maintain the benefit of regular physical activity is needed.

Smoking subjects are at a high risk of developing the metabolic syndrome. Interventional studies offers support to smokers willing to quit through physical activity promotion and healthy diet in order to reduce smoking prevalence whereas avoid weight gain following cessation is a desirable public health goal.

Various public health policies should be implemented in India, in order to control the alarming rise of cardiovascular risks and metabolic syndrome.

Change in the lifestyle is the best way in the prevention of metabolic syndrome.

LIMITATION:

The present study was a basic screening of individuals for assessing the contributors of metabolic syndrome. The other components including tumor necrosis factor - alpha, interleukin-6, fibrinogen, plasma insulin, C–reactive protein and others would have helped in better interpretation.

A cohort study with larger sample size will help in assessing the progression and complications of metabolic syndrome.

Future scope of present study:

The present study reflects the real burden of metabolic syndrome in the special group of people by using specific well defined criteria and laboratory investigations. The individuals who are at high risk with family history of hypertension, diabetes and dyslipidaemia once identified should start medication. Those at low risk are recommended to increase their physical activity and to have high fiber diet. The need of the hour would be to increase the awareness of etiological factors for metabolic syndrome among the general public.

In our population, it is essential to reduce the alarmingly increasing burden of metabolic syndrome by appropriate preventive measures. More frequent assessments will be appropriate for follow up of

this study which could reveal better changes in various components of metabolic syndrome.

For high risk patients in cross sectional population and in some specific category to identify the prevalence of metabolic syndrome, it is necessary to conduct more prospective studies in other regions of our country.

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PROFORMA

Name: Age:

Sex: Occupation:

H/O smoking:

No of cigarettes/day: For how many years:

H/O alcohol intake:

Nil/occasionally/weekly/monthly. For how many years:

Regular exercise:

Time spent/day: For how many years:

Diet: Vegetarian/Non vegetarian.

Duration of sleep/day:

Stress score as per PSS 10:

Duration of service:

Working hours/day:

	Duration	On treatment
H/O Diabetes:		
H/O Hypertension:		
H/O Hyperlipidemia:		

H/O Angina:

Family H/O Diabetes/Hypertension/Hyperlipedemia:

Medications if any:

Anthropometric measurements:

Ht (cm): Wt (kg): BMI(kg/m^2):

Waist circumference (cm): Waist hip ratio:

General Examination:

Consciousness: Orientation:

Comfortable at rest: Pallor:

Cyanosis: Clubbing:

Jaundice: Pedal oedema:

Thoracic/Spine deformity:

JVP: Generalized Lymphadenopathy:

Temperature (^{0}F): Respiratory rate/min:

Pulse rate/min: Blood pressure (mmHg):

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM :

ABDOMEN :

CENTRAL NERVOUS SYSTEM:

BIOCHEMICAL MEASUREMENTS:

Fasting blood glucose level (mg/dl):

Serum triglycerides (mg/dl):

Serum HDL (mg/dl):

மருத்துவப் பரிசோதனை முறைகளைப்பற்றி மருத்துவரிடம் தெரிந்து கொண்டேன். இதனை மேற்கொள்ள நான் முழுமனதுடன் சம்மதிக்கிறேன்.

Perceived Stress Scale – 10

0ஒருபோதும் இல்லை 1அனேகமாக இல்லை
2சிலவேளைகளில் 3பொதுவாகஅடிக்கடி4மிகஅடிக்கடி

1. கடந்தமாதம் ஏதிர்பாராதவிதமாகநடந்தசம்பவங்களால் எத்தனைதடவைகள் கவலையுற்றீர்கள்?

   [0] [1] [2] [3] [4]

2. கடந்தமாதம் உங்கள் வாழ்க்கையிலுள்ளமுக்கியமானவிசயங்களைஉங்களால் கட்டுப்படுத்தமுடியாமல் இருந்ததாகஎத்தனைதடவைகள் உணர்ந்தீர்கள்?

   [0] [1] [2] [3] [4]

3. கடந்தமாதம் எத்தனை தடவைகள் படபடப்புடன் கூடியமனஉளைச்சலைஉணர்ந்தீர்கள்?

   [0] [1] [2] [3] [4]

4. கடந்தமாதம் உங்கள் தனிப்பட்டபிரச்சினைகளைச் சமாளிக்கஉங்களுக்குமனத்தைரியம் உள்ளதெனஎத்தனைதடவைகள் உணர்ந்தீர்கள்?

   [0] [1] [2] [3] [4]

5. கடந்தமாதம் நீங்கள் நினைத்ததுபோல் செயல்கள் யாவும் நடைபெற்றனஎனஎத்தனைதடவைகள் உணர்ந்தீர்கள்?

   [0] [1] [2] [3] [4]

6. கடந்தமாதம் உங்களால் செய்யப்படவேண்டியிருந்தஎல்லாசெயல்களையும் உங்களால் ஈடுகொடுக்கமுடியாமல் போய்விட்டதாகஎத்தனைதடவைகள் உணர்ந்தீர்கள்?

   [0] [1] [2] [3] [4]

7. கடந்தமாதம் எத்தனைதடவைகள் உங்களக்குளரிச்சலூட்டியசெயல்களைகட்டுப்படுத்தக்கூடியதாக இருந்தது?

   [0] [1] [2] [3] [4]

8. கடந்தமாதம் எத்தனைபிரச்சினைக்குரியசெயல்களைமுறியடித்ததாகஉணர்ந்தீர்கள்?

   [0] [1] [2] [3] [4]

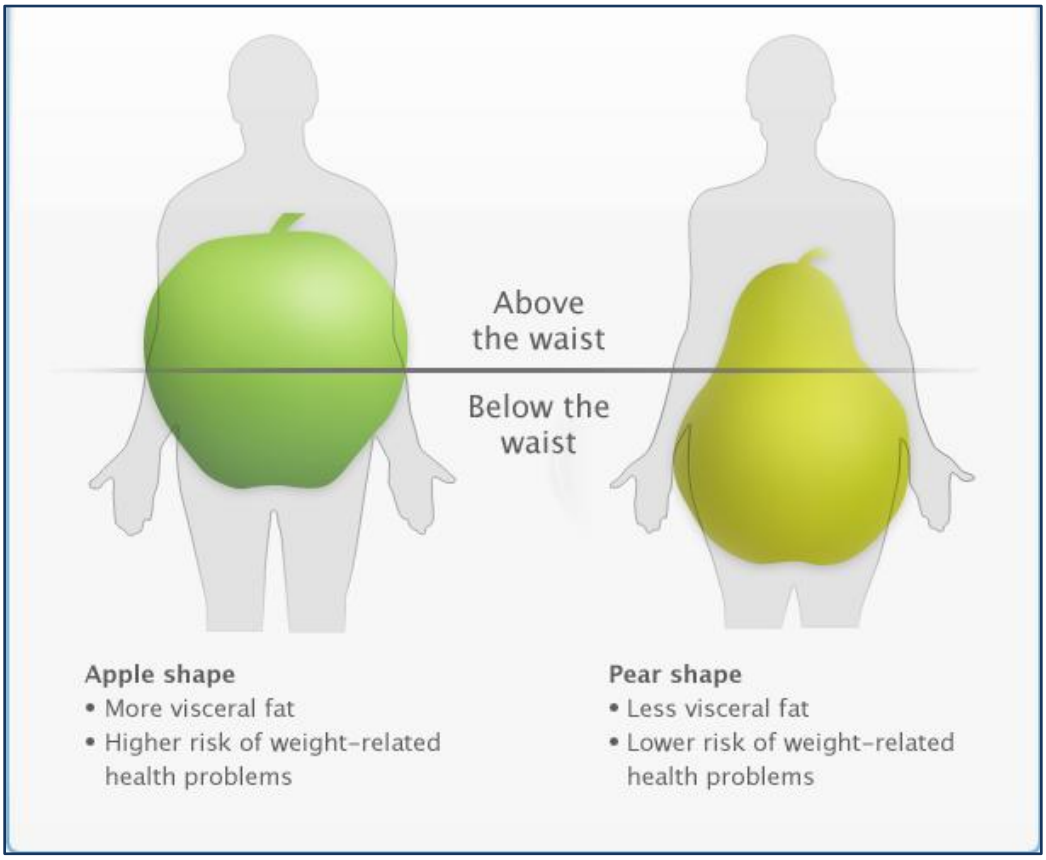
9. கடந்தமாதம் உங்களின் கட்டுப்பாட்டிற்குஅப்பாற்பட்டசெயல்களால் எத்தனைதடவைகள் கோபப்பட்டீர்கள்?

   [0] [1] [2] [3] [4]

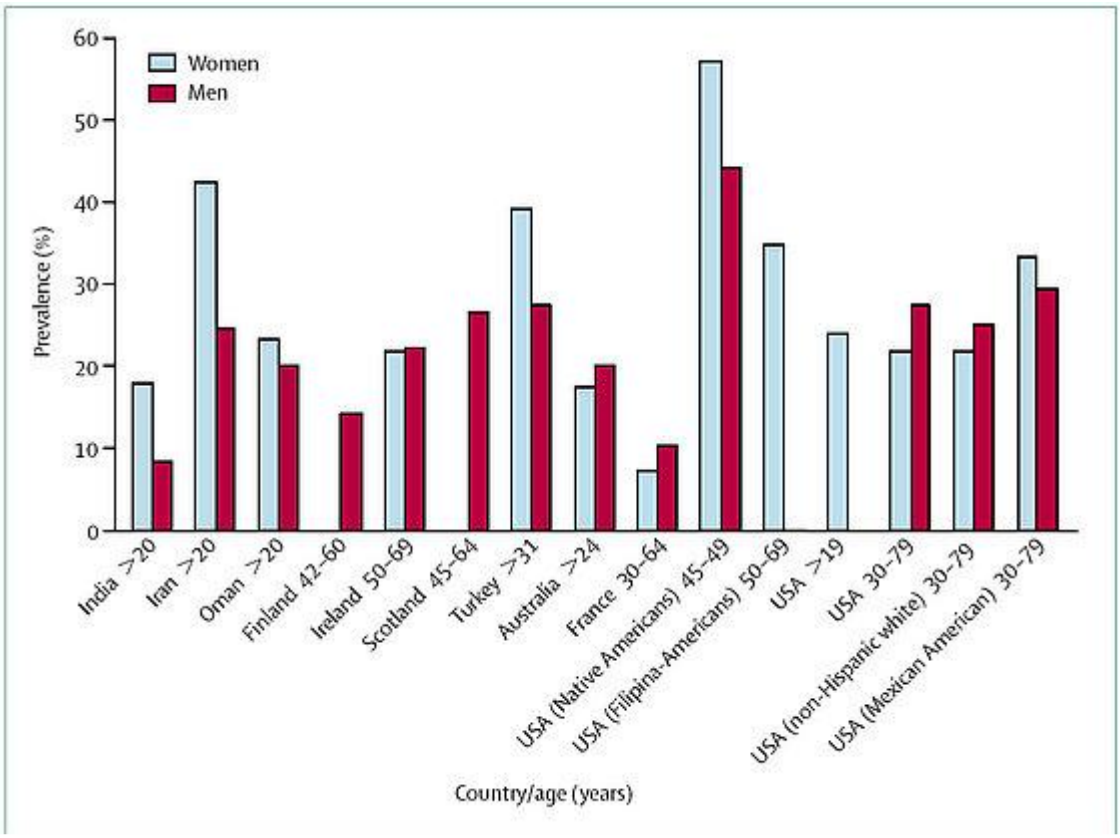
10. கடந்தமாதம் உங்களால் வெற்றிகொள்ளமுடியாதுமென்மேலும் உயர்ந்துகொண்டிருந்தபிரச்சினைகளால் கஷ்டப்பட்டதாகஎத்தனைதடவைகள் உணர்ந்தீர்கள்?

    [0] [1] [2] [3] [4]

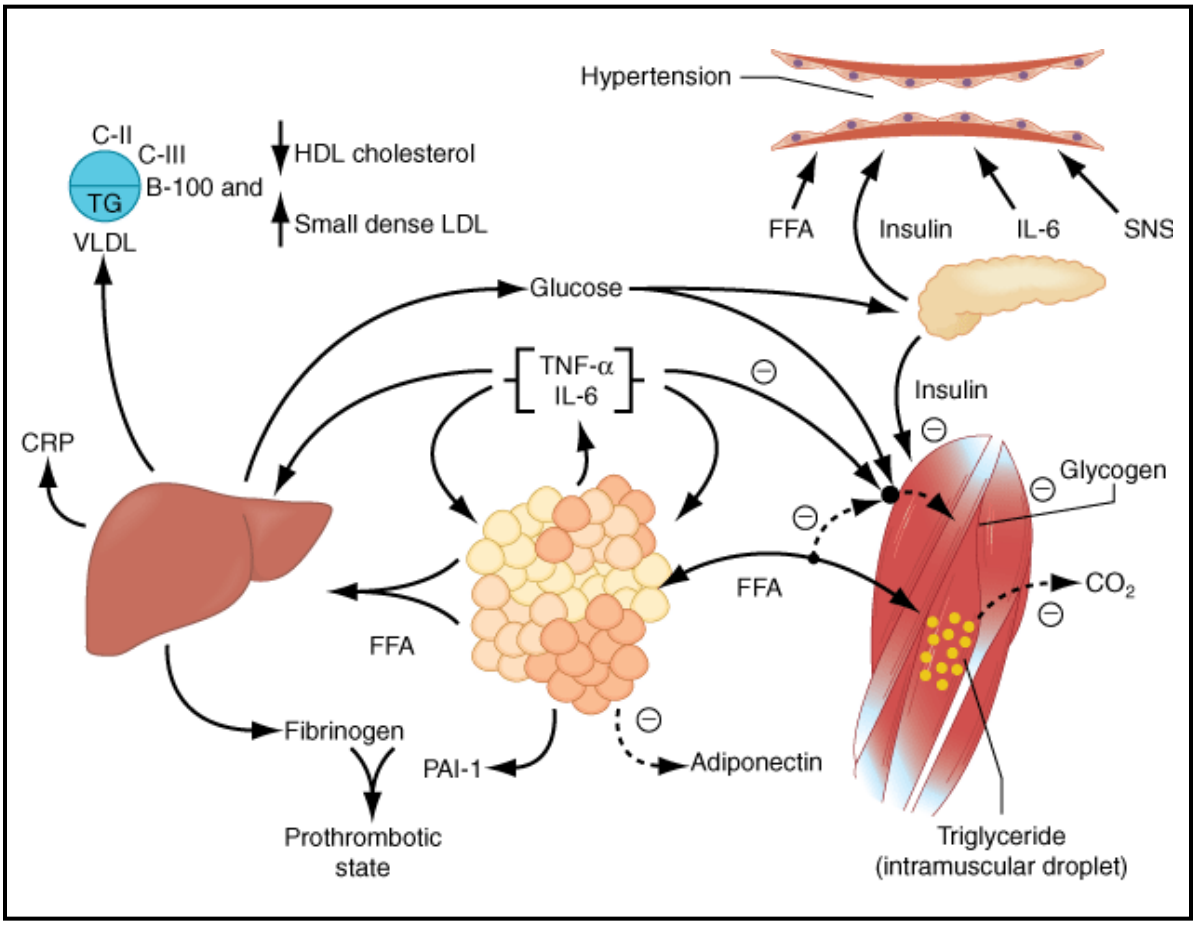
Apple shape Vs Pear shape

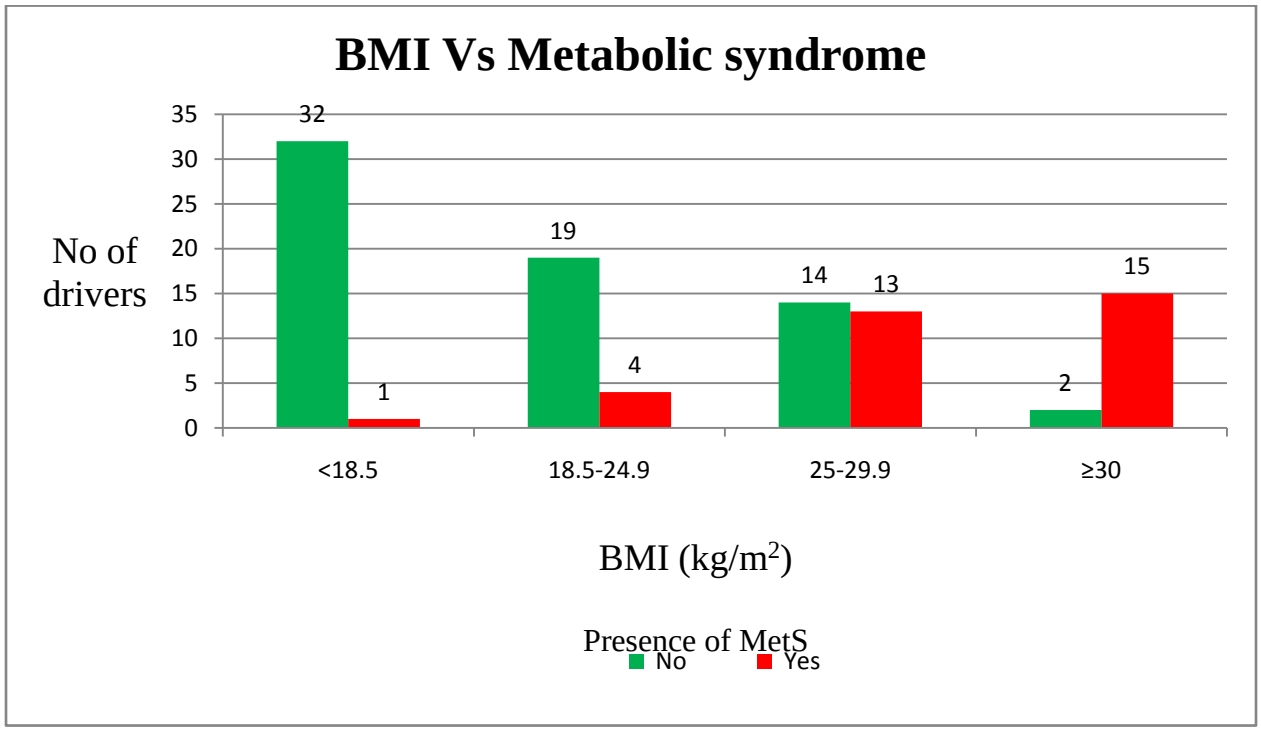


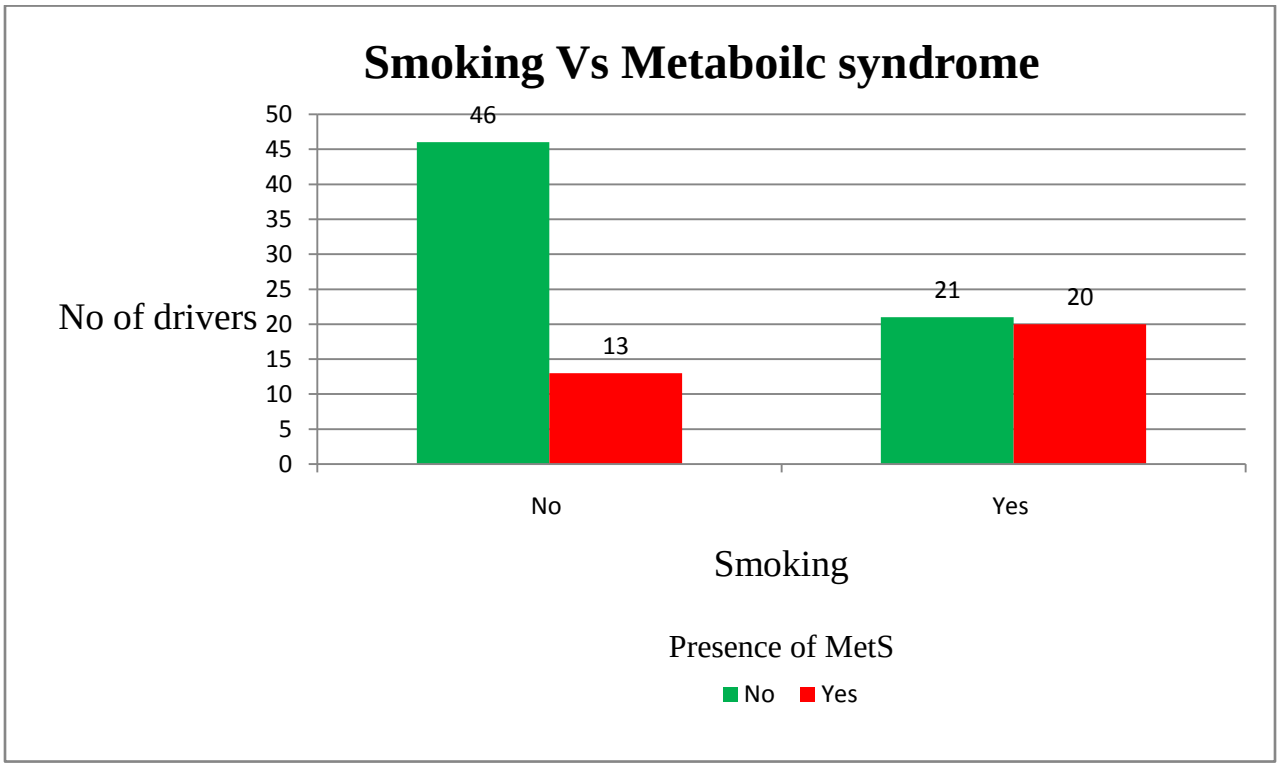
Prevalence of the metabolic syndrome from ATP III definition

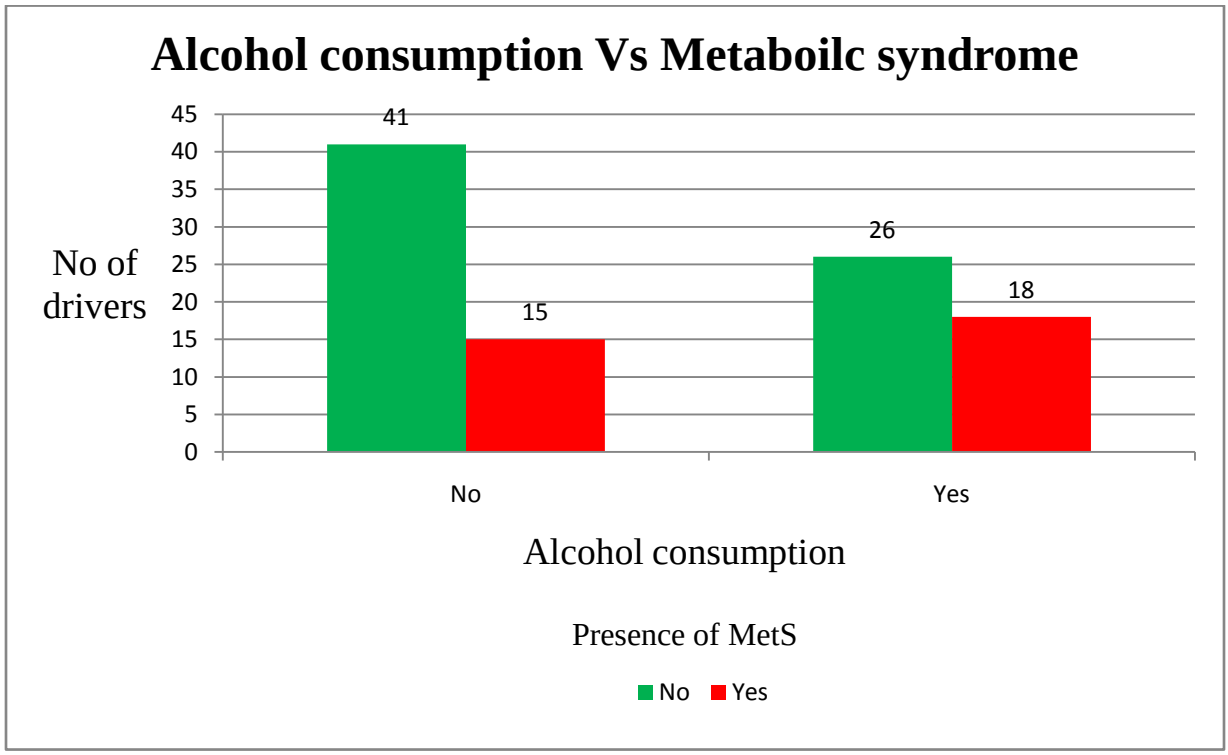


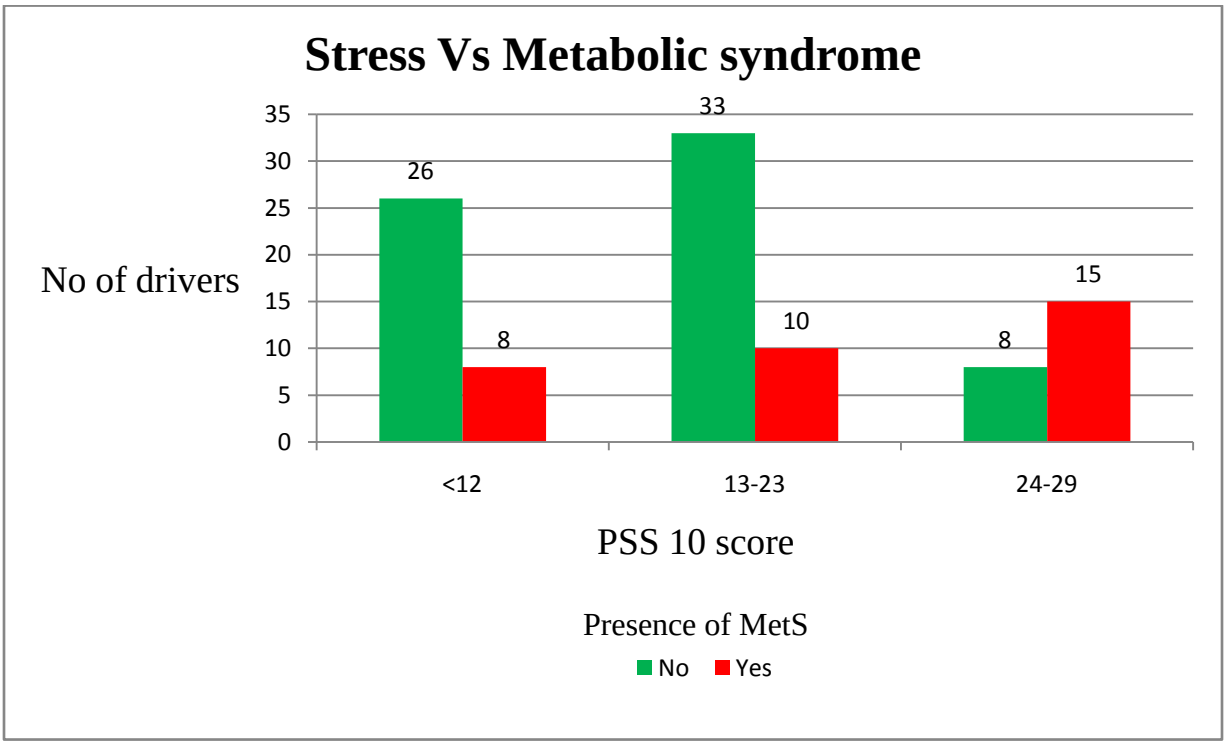
Pathophysiology

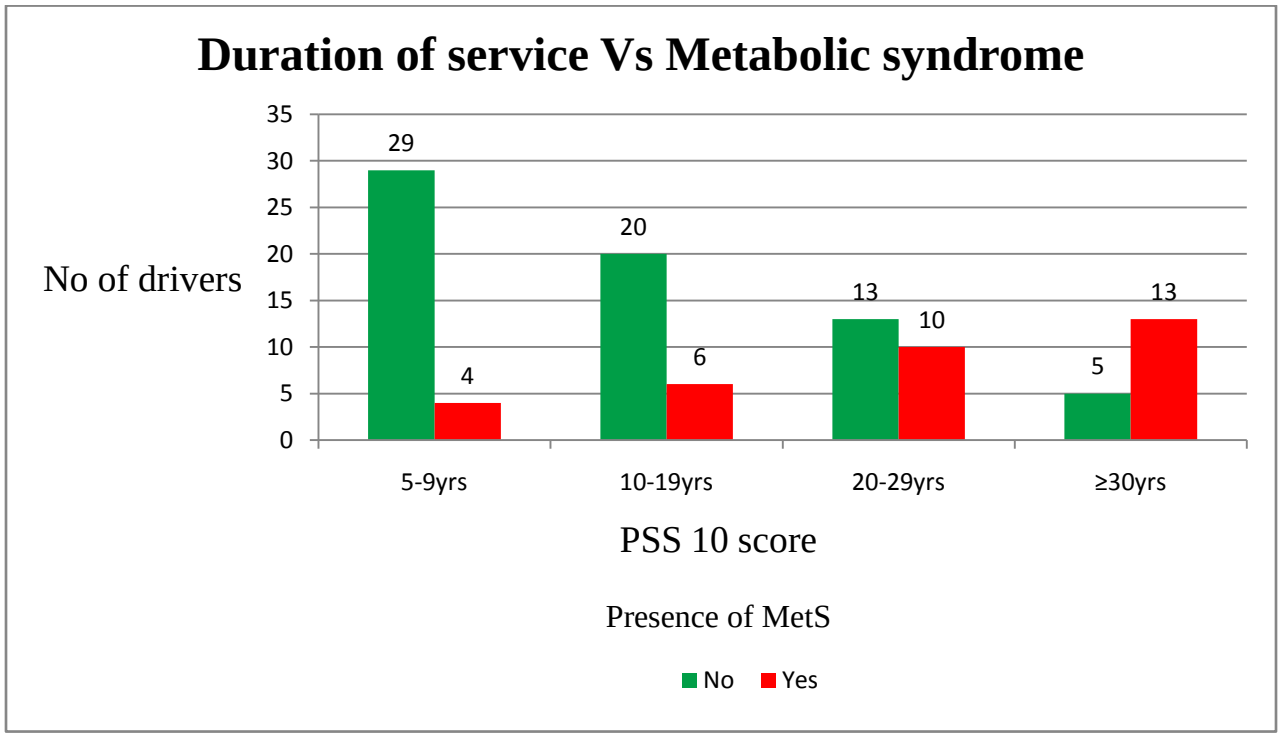



BMI Vs Metabolic syndrome
No of drivers
35
30
25
20
15
10
5
0
32
1
19
4
14
13
2
15
<18.5
18.5-24.9
25-29.9
≥30
BMI (kg/m2)
Presence of MetS
No
Yes


Smoking Vs Metaboilc syndrome
No of drivers
50
45
40
35
30
25
20
15
10
5
0
46
13
21
20
No
Yes
Smoking
Presence of MetS
No
Yes


Alcohol consumption Vs Metaboilc syndrome
No of drivers
45
40
35
30
25
20
15
10
5
0
41
15
26
18
No
Yes
Alcohol consumption
Presence of MetS
No
Yes


Stress Vs Metabolic syndrome
No of drivers
35
30
25
20
15
10
5
0
26
8
33
10
8
15
<12
13-23
24-29
PSS 10 score
Presence of MetS
No
Yes


Duration of service Vs Metabolic syndrome
No of drivers
35
30
25
20
15
10
5
0
29
4
20
6
13
10
5
13
5-9yrs
10-19yrs
20-29yrs
≥30yrs
PSS 10 score
Presence of MetS
No
Yes

AUTO ANALYSER (ERBA – XL-300)

COLLECTION OF BLOOD SAMPLE

MEASUREMENT OF BLOOD PRESSURE

S. No	Name	Age (yrs)	Gender	Duration of service (yrs)	H/O smoking	H/O Alcohol intake	PSS 10 score	Taking R_X for DM,HT HTG	BMI kg/m^2	Waist cm	B.P mmHg	FBS mg/dl	S.TGL mg/dl	S.HDL mg/dl	Presence of metabolic syndrome
1	Boopathi	33	M	12	No	Yes	29	DM,HT	40.7	126	140/90	96	186	32	Yes
2	Manisekar	58	M	36	No	Yes	25	DM	31.8	98	130/90	232	152	33	Yes
3	Natarajan	46	M	21	Yes	Yes	26	-	32.1	101	140/90	98	105	39	Yes
4	Alagupandi	44	M	12	No	Yes	16	-	17.3	76	120/70	105	142	38	No
5	Anand	48	M	15	No	Yes	22	DM	24.7	83	120/70	121	157	43	No
6	Annamalai	56	M	33	No	Yes	18	DM	17.8	92	120/70	116	75	50	No
7	Antony Raj	52	M	30	No	No	24	-	27	94	120/80	78	87	31	No
8	Arun	37	M	8	Yes	No	23	-	24.6	98	100/70	86	83	36	No
9	Amaiah	48	M	19	Yes	Yes	18	DM	27.7	92	120/70	91	119	51	No
10	Balaji	46	M	11	Yes	No	12	-	18.4	75	120/70	98	93	37	No
11	BalaVenkatesh	38	M	5	Yes	Yes	14	-	23.9	83	120/80	90	110	42	No
12	Balu	45	M	15	Yes	No	6	HT	30.5	101	160/110	130	186	40	Yes
13	Arumugam	52	M	23	Yes	Yes	24	DM,HT	23.3	91	120/80	101	90	24	Yes
14	Chandrasekaran	36	M	16	No	No	26	DM,HT	23.7	101	120/70	123	155	33	Yes
15	Chandra sekar	36	M	8	No	Yes	13	-	22.9	86	130/70	96	129	43	No
16	Chidambaram	55	M	23	No	No	12	HT	26.2	86	140/80	92	194	46	No
17	Albert	36	M	13	No	Yes	8	-	24.7	94	120/70	95	90	27	No
18	George	55	M	22	No	No	9	-	25.4	85	120/80	247	118	36	No
19	Sundaramoorthy	43	M	22	Yes	Yes	11	-	18.3	85	130/90	112	115	36	Yes
20	Thangaraj	30	M	9	No	Yes	25	DM,HT	30.4	108	150/100	124	74	36	Yes
21	Devaraj	35	M	9	No	No	12	DM	26.6	94	120/80	104	106	36	No
22	Kaliayamoorthy	40	M	21	Yes	Yes	14	DM	26	100	120/80	102	76	36	Yes
23	Raja	53	M	33	Yes	No	26	DM,HT	26	99	130/90	115	125	36	Yes
24	TamilSelvan	47	M	25	No	No	26	-	18.3	96	110/70	123	92	44	No
25	Ganesan	58	M	25	Yes	Yes	27	DM	30.4	108	130/80	126	94	30	Yes
26	Janakiraman	46	M	18	Yes	Yes	8	HT	18.2	85	110/80	103	117	45	No
27	PalaniSamy	54	M	32	Yes	Yes	19	DM	27.7	101	120/80	109	298	33	Yes
28	Devapragasam	49	M	27	No	Yes	23	-	30.3	109	130/90	89	135	27	Yes

S. No	Name	Age (yrs)	Gender	Duration of service (yrs)	H/O smoking	H/O Alcohol intake	PSS 10 score	Taking R_X for DM,HT HTG	BMI kg/m^2	Waist cm	B.P mmHg	FBS mg/dl	S.TGL mg/dl	S.HDL mg/dl	Presence of metabolic syndrome
29	Doss	40	M	18	Yes	No	19	HT,HTG	27.1	100	140/80	278	94	24	Yes
30	Mageshwaran	50	M	24	No	Yes	20	DM,HT	26.4	92	120/80	85	98	36	No
31	Arivalagan	46	M	16	No	Yes	13	-	17.2	72	100/70	88	87	28	No
32	Chandramohan	45	M	13	Yes	Yes	28	DM	21.6	90	130/80	103	180	35	Yes
33	Chandran	41	M	7	No	No	21	-	18.1	96	120/70	82	96	36	No
34	PasilAhamed	51	M	32	Yes	No	29	-	30	95	140/90	134	127	48	Yes
35	Bala Raman	30	M	7	No	Yes	11	-	18.4	80	120/80	125	159	52	No
36	Kannan	53	M	31	No	Yes	28	-	23.3	94	130/90	98	110	40	No
37	Kannan	32	M	5	Yes	No	18	-	18.3	97	130/90	91	126	45	No
38	Kumar	41	M	17	Yes	No	16	-	23.2	87	120/80	96	89	40	No
39	Muruganantham	56	M	35	No	Yes	12	HT	25.4	95	140/80	70	84	24	Yes
40	Murugesan	47	M	11	No	No	28	-	31.4	105	130/90	86	86	45	No
41	Pandian	54	M	32	No	No	14	HTG	20.5	88	120/80	71	123	39	No
42	Paul Raj	58	M	25	No	No	23	-	18.4	86	130/70	96	129	43	No
43	Radha Krishnan	57	M	35	No	No	21	DM	31.4	99	150/100	116	128	30	Yes
44	Raman	36	M	5	No	No	22	-	22.6	98	120/70	77	90	44	No
45	Srikanth	55	M	26	No	No	10	-	26	97	120/70	88	114	51	No
46	Murali	36	M	8	Yes	Yes	10	-	18	77	100/70	133	100	45	No
47	Muthu Kumar	39	M	9	Yes	Yes	9	-	29.3	114	120/80	97	76	39	No
48	ChellaPandian	38	M	9	No	No	8	-	24.1	103	120/80	83	143	30	No
49	David	30	M	8	Yes	Yes	6	-	18.3	85	130/90	100	117	43	No
50	Gajendran	43	M	16	Yes	No	24	-	17.7	88	140/90	126	138	42	No
51	Raja	31	M	7	No	Yes	15	DM,HT	17.5	85	110/80	103	117	45	No
52	Ramachandran	37	M	8	No	No	9	-	26.3	99	120/80	109	80	33	Yes
53	Sadhasivam	47	M	11	Yes	Yes	17	HT	23.2	88	130/70	105	144	54	No
54	Subash	46	M	10	No	No	28	-	17.9	86	130/70	96	129	43	No
55	Nagarajan	33	M	6	No	No	23	-	16.9	90	120/80	138	132	46	No
56	Babu	55	M	20	No	No	7	-	31	102	110/70	146	108	30	Yes

S. No	Name	Age (yrs)	Gender	Duration of service (yrs)	H/O smoking	H/O Alcohol intake	PSS 10 score	Taking R_X for DM,HT HTG	BMI kg/m^2	Waist cm	B.P mmHg	FBS mg/dl	S.TGL mg/dl	S.HDL mg/dl	Presence of metabolic syndrome
57	Devaraj	35	M	7	Yes	Yes	27	-	24.4	80	120/80	125	159	52	No
58	Kumar	41	M	21	Yes	No	24	DM	29.8	103	160/110	138	92	39	Yes
59	Kumaresan	49	M	13	No	Yes	8	-	18.4	85	120/70	91	133	27	No
60	Mani	39	M	11	Yes	Yes	14	HT,HTG	19.5	92	140/90	103	82	42	Yes
61	Manickam	51	M	31	No	Yes	9	-	17	85	120/70	277	106	42	No
62	Bakiyanathan	58	M	31	No	No	24	DM,HT	27	95	140/100	129	153	33	Yes
63	Radha Krishnan	50	M	24	Yes	Yes	12	-	26.6	91	130/90	96	139	46	No
64	Radha Krishnan	37	M	6	No	No	16	-	18.4	75	140/90	116	130	46	No
65	Selvaraj	37	M	5	No	No	11	HT	23.2	88	130/70	105	144	54	No
66	Soundararajan	52	M	22	Yes	Yes	14	DM,HT	25.4	98	140/90	95	393	45	Yes
67	Palanisamy	52	M	30	Yes	No	25	HT	30.6	96	130/90	71	126	33	Yes
68	Palanisamy	36	M	7	Yes	No	15	-	17.9	91	120/70	87	141	36	No
69	PaneerPandi	30	M	6	Yes	No	22	-	25.3	97	100/70	80	63	29	No
70	Perumal	39	M	28	Yes	No	17	DM	18.3	79	120/70	92	75	37	No
71	Chandramohan	52	M	32	Yes	Yes	20	DM	28.7	90	100/90	187	94	36	Yes
72	Devaraj	56	M	28	No	No	7	-	18	92	100/70	96	103	30	No
73	Govindammal	35	M	7	Yes	Yes	9	-	30.3	112	130/90	114	79	30	Yes
74	Jothimanickam	31	M	6	No	Yes	17	HT	28.7	85	110/70	98	144	27	No
75	Manickan	33	M	9	No	Yes	7	-	17.6	75	140/90	116	130	46	No
76	Dhandabani	46	M	31	Yes	No	10	-	31.3	108	130/80	141	132	33	Yes
77	Selvaraj	44	M	18	No	No	23	HTG	20.5	88	120/80	71	123	39	No
78	Balakrishnan	55	M	35	No	Yes	16	-	35.7	117	120/90	125	38	36	Yes
79	Chandran	47	M	25	Yes	Yes	10	DM	26.8	97	120/80	143	123	21	Yes
80	Edwin raj	30	M	9	No	Yes	9	-	18.3	76	120/70	105	142	38	No
81	Lakshmanan	40	M	13	No	No	14	-	24.7	94	120/70	95	90	27	No
82	Muthukumar	44	M	14	No	No	13	HT	27.7	98	130/90	94	114	42	No
83	Nagarajan	34	M	7	No	No	17	DM,HT	30.4	98	100/70	114	243	33	Yes
84	Padmanaban	39	M	8	No	No	12	HT	23.2	88	120/70	105	120	54	No

S. No	Name	Age (yrs)	Gender	Duration of service (yrs)	H/O smoking	H/O Alcohol intake	PSS 10 score	Taking R_X for DM,HT HTG	BMI kg/m^2	Waist cm	B.P mmHg	FBS mg/dl	S.TGL mg/dl	S.HDL mg/dl	Presence of metabolic syndrome
85	Thangarajan	50	M	23	No	No	6	-	25	96	110/80	103	86	38	No
86	Sasikumar	38	M	6	No	Yes	15	-	18.2	91	120/70	89	117	40	No
87	Sethuraman	42	M	11	No	No	29	-	24.7	94	130/90	91	98	41	No
88	Sheik mohammed	34	M	9	No	No	13	-	17.6	98	120/80	105	138	50	No
89	Srinivasan	32	M	9	No	No	12	DM,HT	18.4	88	110/70	90	130	48	No
90	Kumar	58	M	33	No	No	27	-	26.4	90	130/90	120	126	36	Yes
91	Sekar	48	M	28	No	No	18	-	34.8	107	190/120	76	113	45	No
92	Tamil Selvan	57	M	13	Yes	No	20	-	21.7	89	110/80	109	130	40	No
93	Asokan	53	M	27	No	No	9	-	18.2	84	120/80	83	102	42	No
94	Balagopalan	50	M	31	Yes	No	28	DM	25.2	90	110/70	212	369	48	Yes
95	DavidRajan	30	M	7	Yes	Yes	15	-	17.9	91	100/70	96	157	46	No
96	Deepan	45	M	17	No	No	11	-	24.3	86	120/80	79	78	30	No
97	Ganesamoorthy	45	M	13	No	No	19	-	18.1	84	120/80	83	102	42	No
98	Nagendran	54	M	26	Yes	No	17	-	17.6	94	120/80	109	125	38	No
99	Velmurugan	31	M	5	No	No	12	-	27.6	95	120/80	90	91	32	No
100	Nirmal	39	M	8	Yes	No	10	DM	18	76	120/70	105	142	38	No

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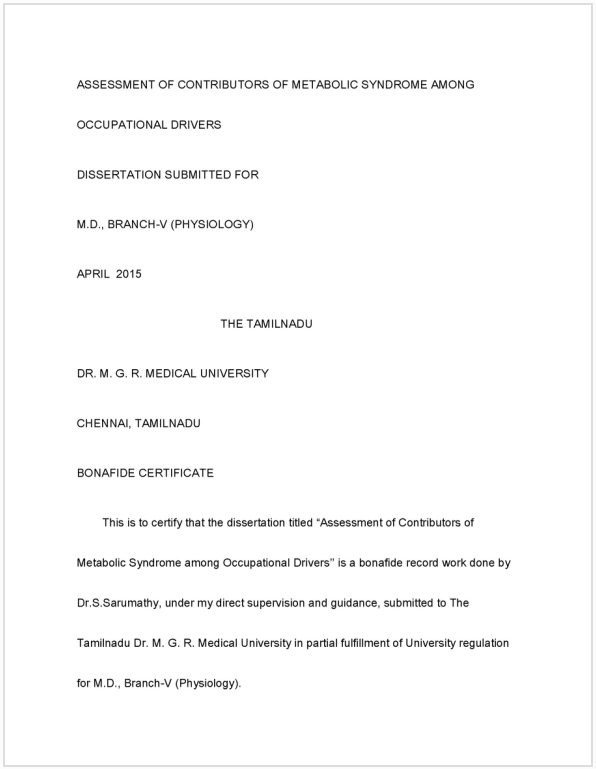

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OCCUPATIONAL DRIVERS

DISSERTATION SUBMITTED FOR

M.D., BRANCH-V (PHYSIOLOGY)

APRIL 2015

THE TAMILNADU

DR. M. G. R. MEDICAL UNIVERSITY

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This is to certify that the dissertation titled "Assessment of Contributors of

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